RHEUMATIC MANIFESTATIONS IN CHILDHOOD AND ADOLESCENCE.

THESIS

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PAUL STEPHANUS MEIRING

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CHILDHOOD AND
RHEUMATIC MANIFESTATIONS IN ADOLESCENCE.

INTRODUCTION.

The importance of this subject can hardly be overestimated when one realizes that rheumatism in childhood is responsible for nearly all the cardiac disease in early life and a considerable proportion in adult life, and that cardiac disease is responsbile for a very large share of the death rate of this country. In 1921 the Registrar General found that 11.7~~6~~% of deaths at all ages and from all causes in England and Wales were due to heart disease and this did not include diseases of the blood vessels.

The frequency of the condition is not fully realized, probably because its minor symptoms owing to their apparent triviality are often ignored or ascribed to something else.

The traditional belief amongst the general public that children cannot have rheumatism dies hard and often prevents an early diagnosis being made. Only too often does one first see these cases when irreparable damage has already been done to the heart.

These considerations, together with the varied clinical manifestations, the unknown etiology and the difficulties of treatment are in the author's

at work give him ample scope for studying the disease in its various guises and for ferreting out possible predisposing causes and contributing factors in the etiology.

In the following thesis an attempt will be made to put together a few observations on the condition as met with in general practice, and to bring these phenomena into their appropriate relation with one's view of rheumatism as a whole.

HISTORICAL OUTLINE.

In reviewing the history of rheumatism one finds that it is a subject of which the closer study has been very much neglected until comparatively recent years.

Thus it was not until towards the end of the 19th century that medical men began to realize that it was not conducive to good treatment to group a host of different joint conditions together and label them "rheumatism". Although rheumatoid arthritis was held to be a disease distinct from rheumatic fever, gonorrhoeal and other forms of synovitis were only beginning to be recognised as separate conditions.

One of the earliest theories advanced was the chemical or Humoral theory which supposed that certain substances, the products of imperfect metabolism, were the causative agents. Lactic acid was first blamed.[1.] Richardson and Rauch succeeded in producing endocardial and pericardial lesions with lactic acid, but later observers did not confirm their results. Later Uric Acid was blamed, particul-[1.]-arly by Haig, but again the evidence was not satisfactory.

From the earliest time damp and cold have been under suspicion. [1]Cullen believed that the disease was due to the direct effect of cold on the joints which were specially liable to attack because of their thin covering, and that the disease started and spread from here.

[1.] J.K. Mitchell in 1831 advanced the theory that chill and exposure irritated the sensory nerves over a large area causing widespread central disturbance which is in turn reflected to the nerves of the various organs and tissues, thus producing the rheumatic manifestations.

More than a century ago [1]Saunders foreshadowed the Microbic theory by suggesting that rheumatism might bear some relation to Malaria, which of course has since been completely disproved. But it was not until later that organisms were actually sought for and discovered.

[2.] In 1886 Dr.A. Mantle described a diplococcus in the blood of cases of Rheumatic Fever and advanced the theory that the disease was of microbic origin. In 1892 [2]P. Guttman isolated the staphylococcus pyogenes flavus from kidney abscesses and pericarded effusion of a case of very acute

rheumatism with pericarditis complicated by abscesses in the kidneys and muscles. He, however, did not consider it a case of true rheumatism, and did not claim this organism as the sole cause of rheumatic fever.

In the same year [2.] Sahli discovered the staphylococcus Pyogenes Citreus in the synovial membrane of the joints in a case of rheumatic fever and also in the pericardial exudation and considered that a staphylococcal infection was the cause of rheumatic fever.

In 1893 [2.] Lanz, experimenting in suppurative polyarthritis, isolated a bacillus from the pus of a brain abscess operated on by Kocher, and called it bacillus pyogenes foetidus liquefaciens. With this he reproduced suppurative polyarthritis in a rabbit.

In 1894 [2.] Maragliano obtained a diplococcus and a staphylococcus from a case commencing as rheumatic fever and ending in Septicaemia.

[2.] Singer at about the same time found staphylococcus pyogenes aureus present in the urine of several cases suffering from rheumatic fever, and formed the opinion that the disease was an attenuated form of pyaemia due to various staphylococci and streptococci.

In this year also [2.] Dana isolated a diplococcus from the meninges of the brain and spinal cord of a case of Chorea following rheumatism, and [2.] Sacaze suggested that a slight insignificant external wound might be the site of infection in rheumatic fever.

In 1896 [2.] Buss of Bremen came to the conclusion that the throat and intestines are in many cases the portal of entrance of rheumatic infection.

[2.] Aschalme in his investigations , 1891-1897 grew a bacillus resembling the b. anthrax anaerobic--ally from the blood of patients who had died of rheumatic fever. The organism when injected into animals, however, failed to reproduce symptoms of rheumatic fever, but caused fatal septicaemia in some cases. His results were confirmed by [2.] Thiroloix who found the bacillus in five cases of rheumatism and was able to reproduce the entire picture of rheumatic fever in animals by inoculation.

In 1897 [2.] Triboulet and Coyon found a diplococcus in five cases in two of which the diplococcus was associated with Aschalme's bacillus, and they concluded that severe cases were due to the two organisms together while milder infections were due to the diplococcus alone.

In the same year [2.] Riva isolated specific organisms from eight cases of rheumatic fever, which varied much in shape in different media and assumed both coccal and bacillary forms.

In 1898 [2.] Apert and Triboulet isolated a diplococcus from the blood of a case of rheumatic fever which produced endocarditis pericarditis and pleurisy in a rabbit, but no arthritis. The organism was identical with that found in eleven other cases. Triboulet considered this organism to be specific.

In 1899 [2.] Westphal and Wassermann cultivated from the heart blood, pericardial effusion, mitral valve, spleen and brain of a case that had died of severe cherea following acute rheumatism, a diplococcus which produced fever and multiple arthritis in eighty rabbits. Cultures of the organism from the joints of the rabbits reproduced the disease in other animals.

Poynton and Payne [2.] starting their investigations in 1900 first turned their attention to finding Aschalme's bacillus, but isolated instead the diplococcus previously discovered by Triboulet in 1898 and by Westphal and Wassermann in 1899.

This organism they studied much more thoroughly than the previous observers. They isolated it in a large number of cases, recovering it from the blood, pericardial fluid, granulations

from diseased valves, rheumatic nodules, tonsils of patients suffering from rheumatic tonsillitis. They cultivated the organisms in pure culture and inoculation in susceptible animals produced poly--arthritis, valvulitis, pericarditis, myocarditis and even choreic spasm. These results were confirmed by Beaton, Ainley, Walker, Beattie and others.

Drs. Coombs and Poynton in their report (Brit. Med. Journal Suppmt., July 3rd 1926, p. 15) on the investigations into the bacteriology of rheumatism since 1906 concluded that there was no rival to the streptococcic theory of acute rheumatism.

This theory is now accepted by the great bulk of medical opinion, but there are still some authorities who believe that the case for the diplococcus rheumaticus has not been proven.

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ETIOLOGY.

Whatever difference of opinion there may be with regard to the identity of the specific organism of acute rheumatism, most authorities now agree that the cause is microbic. In support of this view a considerable amount of evidence has accumulated within recent years. Thus in 1895 [3.] Newsholme proved the tendency of the disease to occur in irregular epidemics by a large amount of statistical evidence. He pointed out that epidemic waves occur every 3 - 6 years, and showed that the rates of mortality and frequency of the disease vary in a similar manner to those of such infectious diseases as Scarlet Fever and Erysipelas. He considers acute rheumatism to be a soil disease due to a saprophytic organism which is drowned out in wet years, and multiplies rapidly in dry years, thus accounting for the increased prevalence in these years. [4.] Hirsch could find no constant meteorological influences to account for local epidemics, and concluded that the disease was one of the acute infective diseases.

Of considerable interest is the occurrence of the so-called "house epidemics", a large number of which have been recorded. Thus Hawthorne in

B.M.J. December 26th, 1903, gave particulars of five cases occurring in the same family in or near one house at intervals of a few days.

This, however, may be taken as evidence either of an hereditary predisposition or of the infective nature of acute rheumatism. Of more significance is the fact that members of different families living in succession in a particular house have acquired the disease. Thus Dr. Reginald Miller[5.] reports the development of rheumatism in turn in the children of three families occupying in succession the same basement. House and local epidemics also raise the possibility that rheumatism may not only be infective but also infectious. I have recently attended at the same time two children in bed in the same house and of the same family, one suffering from chorea and arthritis, the other from tonsillitis and arthritis. Neither of the parents had ever suffered from acute rheumatism. In another family while I was attending a girl of 7 years for a severe attack of chorea with endocarditis and arthritis, her brother of 5 years developed a mild attack of chorea with slight arthritis. Here, too, hereditary predisposition did not play a part.

The seasonal incidence of acute rheumatism closely resembles that of Scarlet Fever and as in

many acute infectious diseases certain families and persons show a special proclivity to the disease. The symptoms, as pointed out by Osler[6], show a striking similarity to those of the acute infections, especially pyaemia; thus leucocytosis, sweating irregular fever, a tendency to involvement of the serous membranes, and endocarditis are features shared by both conditions.

While there is general acceptance of the microbic theory, there is still considerable difference of opinion among authorities as to what the causal organism is. Results obtained by various bacteriologists have been very discordant, and consequently several different hypotheses have been advanced. These I propose to discuss in turn:-

1. Rheumatic Fever is the result of an infection with a Specific Anaerobic bacillus (Achalme). This organism, however, has only been found in a small number of cases and then chiefly after death. It has also been found in a case of Malaria. The results produced by inoculation into animals have not been convincing. Achalme himself was able to produce only a form of malignant oedema with this organism, Thiroloix being the only observer whose results resembled acute rheumatism. Dr. Hewlett has pointed out that Achalme's bacillus is probably identical with B. enteridis sporogenes. The bacillus

has been found in association with other organisms including B. Coli in cases of acute rheumatism, and this is looked upon as significant of a terminal infection or post mortem invasion.

2. Rheumatic Fever is an attenuated form of pyaemia caused by staphylococci and streptococci (G. Singer). There is very little evidence to support this claim, his results not having been confirmed by other observers. Also the hypothesis does not account for the absence of suppuration in severe and rapidly fatal cases of rheumatic fever, and attenuated pyogenie cocci do not produce the lesions characteristic of acute rheumatism when injected into animals.

3. Rheumatic Fever is the result of a mixed infection with Achalme's bacillus and diplococci (Triboulet and Coyon).

4. Rheumatic Fever is not a disease in itself but a particular reaction in predisposed persons to various organisms especially streptococci (Menzer).[7] Menzer considers that this view is supported by the fact that streptococci occurring in rheumatic throats act in the same way as those from normal throats. To this [7] Bullock raises the objection that streptococci are not constant in Rheumatic Fever, and there is difference of opinion as to how far the throat lesions are characteristic of acute rheumatism.

Moreover it is very difficult to believe that the very definite clinical picture of acute rheumatism can be caused by many and varied infections.

5. The cause is a specific diplococcus (Poynton and Payne). This is the hypothesis which has gained most general support. It is the only one supported by a substantial amount of experimental evidence and by several independent observers. Poynton and Payne reproduced all the classical lesions of acute rheumatism with their organism and recovered it from all of these lesions. These results were confirmed by Drs. Beaton and Ainley Walker and Beattie.[7.] Later Dr. Ainley Walker and Mr. Ryffel made the interesting observation that relatively large quantities of formic acid were generated in the culture of the diplococcus, and they also found appreciable quantities of formic acid in the urine of rheumatic patients, whereas in normal urine it is absent or present only in a trace. Under Salicylate treatment the amount of formic acid diminished. The origin of the formic acid they attributed to the oxidation of Sarcolactic acid.

There is, however, a formidable array of observers whose results have been chiefly negative.

Among these are [7.] Bullock and Thompson, Pribram, Cole, Phillip, Weichselbaum and Harbitz. From this Bullock infers that the diplococcus is not present in every case of acute rheumatism. He also points out that the bacteriological results in the early stages of rheumatic fever have been mainly negative, and that positive results have been obtained mostly late in the disease and shortly after death. But as suggested by Poynton and Payne, in early cases the micrococci are probably largely destroyed in the tissues and the exudates by leucocytes and phagocytic endothelial cells. Later and in more severe cases, the lining endothelium is damaged and cultures from effusions will yield positive results.

[7.] Menzer in 1902 experimented with streptococci isolated from rheumatic and non-rheumatic throats and obtained identical results from the injections into animals, viz. arthritis, endocarditis, peri--carditis, lung abscesses and local abscesses. Streptococci obtained from mammary pus gave the same results.

[7.] Cole produced arthritis in the rabbit with six strains of streptococci isolated from non-rheumatic lesions. The joint lesions resembled the polyarthritis of rheumatic fever closely and

occasional exacerbations occurred and recovery resulted. In two cases endocarditis also developed. From this Bullock concludes that the results produced by the diplococcus rheumaticus do not differ essentially from those produced by cocci which have been isolated from non-rheumatic lesions. But the multiplicity of organisms in the tonsils rather detract from the value of Menzer's evidence, and the relationship of tonsillitis to acute rheumatism is still disputed.

6. The hypothesis that rheumatic fever is due to a virus still unknown (Pribram, Lenhartz, Bullock and others) although it has a considerable number of adherents, rests chiefly on negative bacteriological results and cannot, therefore, be seriously upheld as an alternative to Poynton and Payne's hypothesis.

7.

That the diplococcus rheumaticus is a cause of acute rheumatism is undoubted, that it is the only cause is probably the case, but there is as yet no certain test to differentiate it from streptococci obtained from non-rheumatic sources other than the results of experimental inoculation into animals.

PREDISPOSING CAUSES.

Although the essential cause of acute rheumatism must be looked upon as microbic, clinical experience teaches us that there are certain subsidiary factors in the etiology of acute rheumatism which have an important bearing on the incidence of the disease.

Age. The disease is essentially one of childhood and early adult life, although it may occur at any age. A case has been described at 23 days and another at 80 years. In my own practice the majority of first attacks have occurred between 5 and 15 years. One case at 3 years and two at 4 years are the youngest I have met with. I cannot recall a single first attack occurring after 20 years. Such cases as I have seen in adults have all been subacute attacks in people who had suffered from one or more of the manifestations of rheumatism as seen in children at some time during childhood or adolescence.

Sex. In childhood the disease is more frequent in females than in males. Of [8] Poynton and Paynes series of 500 cases, 319 were females and 181 males. The proportions, however, differ at different stages during childhood. According to the statistics of the [9] Collective Investigation Committee in the

first period of from 1 to 5 years boys preponderate in the proportion of 5 to 1. From 6 to 10 years they become nearly equal in the proportion of 15 to 14. From 11 to 15 years the incidence in girls increases rapidly so that they suffer in the proportion of nearly 2 to 1. The greater incidence in females between 11 and 15 years is said to be due to their greater liability to attacks of chorea at puberty. Of 31 rheumatic children that I have personally attended 18 were females and 13 males. This preponderance of females also occurred in cases below 10, where 10 were females and 8 males.

Heredity and Family Incidence. On this question statistics are often unreliable. The poorer classes amongst whom acute rheumatism mainly occurs can seldom give a reliable history. They look upon all painful conditions of the joints as rheumatism and apply the same term to numerous other pains in different parts of the body. In this they are rather encouraged by careless practitioners who do not trouble to give an exact diagnosis, labelling as simply "rheumatism" such common conditions as rheumatoid arthritis. The significance of such manifestations of acute rheumatism as growing pains, chorea and rheumatic nodules is not known to the parents and consequently a possible hereditary taint

may be overlooked in the absence of a definite history of articular rheumatism. It is obvious, therefore, that to obtain reliable statistics a very close enquiry into the family history has to be made and access to the other members of the family is desirable. [9]Cheadle found a definite family history of the same affection in near blood relations in 70% of his own private cases and in
10. 30 consecutive cases, 23 giving a family history. The frequency with which one meets with more than one case of acute rheumatism in the same family suggests a family predisposition, but this may on the other hand also be looked upon as evidence in favour of its being a house disease.

This, however, does not account for those cases of rheumatic fever which occur in different members of the same family living in different localities many of which have been reported.

I have under my care now four families in each of which two children in quick succession developed attacks of acute rheumatism in oneor other form. There is some evidence that in rare cases where the mother is actually suffering from rheumatic fever in late pregnancy, the child may be born actually suffering from rheumatic fever, the infection being transmitted directly through the placental circulation. It has also been suggested

that some endocardial lesions found at birth may be due to foetal rheumatism. Acute rheumatism in both parents intensifies the tendency to the disease in their offspring and makes it more severe - thus [11] Cheadle, Goodhart and Garrod found that their worst cases were those who had inherited a double taint from their parents.

Is there a Rheumatic diathesis? There is a certain type of child whose appearance at once suggests the possibility of acute rheumatism. Such a child often looks fragile and anaemic, has a sharp intelligent face and is nervous, excitable and highly strung. Such children when first examined for some trivial ailment are sometimes found to be already suffering from some mild form of acute rheumatism.

Race, Climate and Soil. Acute rheumatism is commoner amongst white races than coloured. In England it is said to be more common among the blonde types than among dark types.

It is found all over the world but is commoner in temperate and subtropical climates. Cold can have but little influence on the incidence of the disease, as it certainly is not most prevalent in the coldest months of the year in this country.

Also Scotland, although the colder country suffers less than England.

On the other hand countries which show wide diurnal variations of temperature, e.g. Cape Colony and Egypt are specially liable to acute rheumatism. Such variations would be more conducive to "chills" which, I think, it is generally agreed, often play an important part in determining the onset of an attack of acute rheumatism. I have been struck by the frequency of a history of a "soaking" a few days or a week or two prior to the onset of an attack in children. A common history obtained from the mother is that a few days before symptoms were noticed the child got wet to the skin going to school and kept the wet clothes on until he came home after school hours.

There seems to be some difference of opinion as to which soil is most favourable for acute rheumatism. Some maintain that cold, damp, low-lying localities are most to blame, others that the disease develops most frequently on dry, hilly, bleak sites. I have seen it with about equal frequency under these different conditions.

[12] Dr. Newsholme believes that the disease is more prevalent when the level of the subsoil

water is low and the earth temperature high, such conditions being favourable to the multiplication of the saprophytic organism he believes to be the cause of acute rheumatism.

Dampness in the houses has always been looked upon with suspicion. In the report of the Sub. Committee on Rheumatic Heart Disease in Children (Brit. Med. Journal Supplmt. July 3rd, 1926, p.7) Miller gives the following statistics on the prevalence of dampness in the homes,, the first that have ao far been compiled on an extensive scale.

Damp in Homes: Results of Investigation in 196 cases.

	No: of Cases	Rooms Damp	Rooms Dry.
West London	114	76	38
Bristol	52	30	22
Bath	30	16	14
	196	122	74

In my own series of cases while I have by no means found it always present, even in the poorest houses, dampness has been sufficiently common to be looked upon as an important predisposing cause. In the case of houses situated on dry hilly sites I have often found the walls on the exposed side of the

house wet. Acute rheumatism often seems to favour particular houses, thus [12.] Friedlander of Leipzig saw twelve cases from the same house in three years, and during another three years eighteen cases from two houses. As such cases cannot be accounted for by hereditary predisposition, where they are of different families, it seems reasonable to suppose that there is some infective agent which finds the conditions in these houses particularly suitable for its development.

Seasonal Incidence. There is general agreement that seasonal changes play a part in the prevalence of acute rheumatism. Fluctuations in the prevalence of the disease are fairly constant for the same locality, but differ when compared with those of other localities. Generally speaking, the disease is more common in the latter half of the year and reaches its maximum in September, October and November. The majority of my own cases have occurred from August to October.

Previous Illnesses. [13.] Poynton and Payne believe that Scarlet Fever, Measles and Diphtheria by causing sore throat predispose to rheumatism, especially in those with an hereditary taint. I

have noticed the frequent occurrence of Scarlet Fever in the previous history of cases of acute rheumatism. Phthisis is said to be not uncommonly associated with attacks of acute rheumatism. I have met with a family in which the mother and two daughters suffered from acute rheumatism while a third daughter had phthisis.

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Pathology.

The rheumatic infection is commonly believed to enter by way of the tonsils. The frequency with which tonsillitis precedes or coincides with an attack of acute rheumatism and the fact that the tonsils are known to be a portal of entrance in other infective conditions at least lend support to this view. On the other hand, first attacks of acute rheumatism occur in many children whose throats apparently differ in no way from the normal healthy throat. It is possible in these cases that some part of the alimentary tract may be to blame.

Recently Miller (Supplt. Brit. Med. Journ. July 3, 1926) from his own investigations of the streptococcal contents of the stools found that this was often excessive in rheumatic cases. He suggested that the excess of streptococci in the bowel acting either as direct infective agents or indirectly by lowering resistance to streptococcal systemic infection might be the explanation of relapses and chronic rheumatism in children.

The actual relationship of tonsillitis to acute rheumatism is still disputed, some believing that tonsillitis is itself caused by the organism of acute rheumatism and is the first evidence of the disease, others that the opened or inflamed crypts of the

tonsils form channels by which the infection enters.

Subsequent attacks of acute rheumatism may be due either to organisms which have been lying latent in the tissues, especially the tonsils, becoming active again or to reinfection. The diplococcus rheumaticus according to [13] Poynton & Payne is probably present in the tonsils of the healthy subject. In certain people, especially those with an hereditary taint, if they are exposed to one or more of the predisposing conditions described above, the diplococci develop pathological properties, gain access to the system and are carried by the blood stream to different parts of the body, where they set up numerous and varied manifestations.

The Subcutaneous Nodule.

This is the typical lesion produced by the rheumatic infection, and allowing for differences in structure of the various organs affected, the changes in it occur in all other rheumatic manifestations in the body. Poynton & Payne have demonstrated the presence of the diplococci in the centre of the nodule which is formed by a homogeneous mass of fibrin and necrotic tissue. Surrounding this there is a zone of leucocytic infiltration and this in turn is surrounded by young fibrous tissue with dilated capillaries.

There is evidence therefore of an infection which is resisted by leucocytic infiltration and tissue reaction. If the resistance proves effective the nodule is absorbed, if ineffective the nodule persists and permanent scar tissue is formed. The majority of these nodules are soon reabsorbed before they reach the stage of fibrosis. A few however, persist as fibrous nodules. These lesions arise mainly from fibrous tissue and may occur in any situation where there is fibrous tissue, but only a few situated over bony prominences and along tendon sheaths are visible. [13]Poynton & Payne suggest that slight trauma in the form of friction and constant movement probably increases the severity of the infection over exposed parts and in the vicinity of joints, hence the nodules are bigger and become visible. In the same way the constant movement of tendons and also the well known preference of the diplococcus for serous membranes explain their comparative frequency in this situation.

Submiliary Nodules.

Occuring in myocardial rheumatism have been described by [13]Aschoff, ~~Farvara~~ Tawara and Carey Coombs. These are microscopic rounded or fusiform areas occurring in the intermuscular connective tissue.

They consist of large spindle-shaped cells about 400 μ in diameter, often multinucleated, surrounded by plasma cells and leucocytes - chiefly mononuclears. The nodules are often associated with an arteriole and may undergo either cloudy swelling or cicatrisation.

The Fascial and Subcutaneous Tissues suffer much from the rheumatic infection. In the former a rheumatic fibrositis is produced. The rheumatic nature of this lesion in any particular case is difficult to prove, but clinically the condition is so commonly associated with other manifestations of rheumatism in children, and such a ~~permanent~~ prominent feature of the subacute rheumatism of older subjects that one cannot doubt the existence of a rheumatic form. In severe cases tender nodules arising from the fascia are sometimes made out in the muscles.

According to [14]Stockman the essential change is sero-fibrinous exudation with proliferation of connective tissue which is ultimately either absorbed or undergoing fibrosis forms permanent fibrous nodules and indurations. The inflammatory changes cause pain on muscular movement and later when fibrosis sets in they interfere with the free action of the muscles, and cause pain by pressure on the

sensory nerves especially after exposure to chill or overstrain.

In the subcutaneous tissue numerous nodules may arise as already described.

Joints.

Although these do not suffer as much in children as in adults, they are nevertheless frequently attacked. Arthritis of all degrees of severity may occur. In very slight attacks the joint may show very little change. In a severe attack there is effusion of blood stained synovial fluid into the joint, and the synovial membrane is swollen and congested. There are small haemorrhages and leucocytic infiltration in the synovial tissue.

The connective tissue of the capsule is swollen and infiltrated with serous exudation. Later the effusion in the joint becomes sero-fibrinous, and a fibrinous deposit is formed on the surface of the synovial membrane. The peri-articular fibrous tissues often suffer severely in children, more so in many cases then the joints themselves, the changes being similar to those occurring in the capsule of the joint. Usually the inflammation subsides in a few days and the effusion is absorbed, but in some cases fibrosis apparently occurs in the peri-articular tissues, resulting in prolonged

stiffness of the joints. Although permanent changes in the joints themselves are infrequent [15] Poynton & Payne believe that individual joints may sometimes remain swollen and painful after the others have subsided, and ultimately pass into a condition of rheumatoid arthritis. I have never seen this transition in practice. It is possible that the local injury produced by the rheumatic organism may act as a suitable basis for operation by some other toxin circulating in the blood, in the same way as continual strain thrown on a joint by a deformity, or traumatic injury of a joint sometimes leads to rheumatoid arthritis.

In early cases of arthritis the organisms are destroyed and cultures of the joint effusions are usually sterile. Later and in severe cases the organism can be isolated from the effusion.

The Heart,

The Myocardium seldom escapes in acute rheumatism. The changes consist in:-

(1) The submiliary nodules in the interstitial tissue as described above.

(2) The muscle fibres undergo fatty degeneration, vacuolation and loss of transverse striation. The increase of interstitial fibrous tissue is usually slight but may be extensive in some recurrent cases.

The fibrous changes may be general or confined to certain areas of the heart wall, or may be found immediately beneath the pericardium.

These changes although they may occur independently are often secondary to pericarditis, and in some cases are associated with endocarditis.

Cardiac nerves may be involved in the areas of fibrosis and the A.V. bundle may be damaged.

The Endocardium.

The organisms are carried to the heart valves - chiefly the mitral and aortic valves the right side of the heart being rarely affected. Having reached the valve they produce necrosis, leucocytic infiltration, cellular proliferation and vascularisation in the sub-endothelial tissue with the formation of small projections from the margins of the cusps. These later break down forming granulations on which fibrin may or may not be deposited. These vegetations heal by fibrosis causing thickening of the valves. The thickened endocardium may become atheromatous and then calcified or atheromatous patches may soften and break down leaving an ulcer. Ultimately the valve segments are much thickened and shrunken causing imperfect closure of the valve and incompetence.

Diplococci are not readily found in the heart

valves in the early stages, because they are rapidly destroyed, but [15]Poynton & Payne have found them in the sub-endothelial tissue and at the base of the valve. Later when granulations have formed they may be found on the surface.

Another type of lesion is found unassociated with an acute attack of rheumatism. This consists of a gradual fibrosis of the valves, chordae tendineae and papillary muscles, causing thickening and shrinkage of the valves the segments of which are adherent at their edges. The valve most usually attacked is the Mitral valve with stenosis as a result, but it may also cause Tricuspid and Aortic Stenosis. This process may follow on an acute attack of endocarditis, but very often it develops insidiously from the commencement and is steadily progressive with little warning as to the state of affairs until definite and irremediable damage has been done.

The Pericardium.

The micrococci produce congestion in the pericardial tissue with exudation of serum, coagulation necrosis, leucocytic infiltration, cellular proliferation and capillary haemorrhages.

The lining endothelium is destroyed and serous sero-fibrinous or sero-purulent exudations take place into the cavity.

The amount of effusion is variable. It may be very slight but in a few cases it is very large. In severe cases great thickening of the pericardium results. Organisation of the exudate causes adhesions between the two layers of the pericardium, and the inflammatory process spreading into the neighbouring mediastinal tissues causes adhesions between the pericardium on the one hand and the chest wall, diaphragm and pleura on the other.

As in endocarditis there is also a slow form consisting in a gradual fibrosis of the pericardium resulting in complete adhesion of the pericardial layers the space between them being entirely obliterated. The firm fibrous sac thus produced greatly interferes with the heart's action, and in childhood interferes with the development of the heart so that in time the body outgrows the strength of the heart which is unable to keep pace with it, and general dropsy eventually follows.

Arteries.

Arteritis may be produced but is exceptional in the young. Embolism however may cause arterial damage.

The Veins.

Phlebitis is rare but occasionally is so severe as to be dangerous to life.

The Blood.

There is nearly always anaemia though seldom very severe. There is always a moderate polymorph leucocytosis. Diplococci have been found in the blood but usually they cannot be found.

The Nervous System.

Although highly irritative the rheumatic infection is not very destructive to nervous tissue. Lesions in the central nervous system fall into two classes:-

(1) The presence of diplococci in the pia mater leads to the development of submiliary nodules near the minute blood vessels. Nodules also occur in the connective tissue supporting the minute blood vessels and more rarely in the neuroglial tissue. In the brain thrombosis, perivascular exudation and small areas of softening may result from the infection.

The meningeal changes on the whole resemble the subcutaneous nodule, and very rarely go on to actual meningitis.

(2) The nerve cells of the brain as a result of toxaemia show changes in their nuclei and slight damage to the medullated nerve fibres.

Parenchymatous and interstitial changes may occur throughout the nervous system giving rise to

neuritic pains as in Rheumatic Sciatica.

Respiratory System.

The Tonsils. The relationship between tonsillitis and acute rheumatism is not definitely settled, partly because the multiplicity of organisms present in the tonsils detracts from the value of bacteriological examinations, and because clinically inflammation of the throat associated with acute rheumatism differs in no way from that occurring in numerous other infective conditions. [15]Poynton & Payne however, who believe that the two conditions are directly associated have found rapid multiplication of strepto-diplococci in 'rheumatic' tonsils and these they believe may give rise to three sets of changes:-

1. Acute general inflammation.
2. Foci of inflammation with follicular exudation.
3. Fibrino-plastic exudation.

After recurrent attacks there is enlargement of the tonsils and foci of disease with necrosis may be found deep in the tonsils.

There is commonly some enlargement of cervical glands associated with the tonsillitis.

Pleurisy is common. It is usually dry but occasionally sufficient sero-fibrinous exudation

is poured out to require tapping. The microscopic changes are similar to those occurring in pericarditis. The diplococcus has been isolated from the effusion.

Lungs. Broncho-pneumonia apparently is occasionally due to the rheumatic infection. Sero-fibrinous exudation takes place. In severe cases there may be small haemorrhages underneath the pleura and haemorrhages into the Alveoli.

The pulmonary oedema which exceptionally occurs in acute rheumatic carditis is probably just the mechanical result of heart failure, and not directly due to the rheumatic infection.

Kidneys. The diplococcus has been isolated from the urine of virulent cases of Acute Rheumatism. Acute Nephritis may occur and also infarctions leading later to areas of fibrosis. A diffuse interstitial fibrosis has been noted in fatal cases of mitral stenosis.

Alimentary Canal.

Nothing is definitely known as to how far the alimentary canal suffers from the rheumatic infection. It is believed by some that appendicitis sometimes is rheumatic. It has been produced in rabbits by inocculation with diplococci obtained from acute

rheumatic lesions in man. Also in a child who had tonsillitis and appendicitis simultaneously the diplococcus was isolated both from the tonsils and the appendix and this organism produced appendicitis, Arthritis and endocarditis when inoculated into rabbits. The evidence is scanty and does not warrant a definite opinion.

Clinical Manifestations.

Rheumatism in children tends to be insidious in its onset. It may make its first appearance in one of many guises. Unfortunately the earliest manifestations are often slight and do not excite alarm in the parents at a stage when much can be done to save the patient from the ravages of the disease. It may first announce itself as a sudden, moderately severe attack of arthritis with or without endocarditis. Such cases are as a rule seen early by the doctor, but more often the onset is insidious showing itself as "growing pains" or very mild attacks of arthritis or fibrositis the significance of which may not be recognised for a considerable time, while cardiac mischief in the shape of endocarditis proceeds apace unnoticed and unchecked.

Any one of the manifestations may occur by itself in the rheumatic child but various combinations of them frequently occur. Of these, arthritis with endocarditis is, in my experience, much the commonest. Arthritis with pericarditis is much less common, but any combination may occur.

In studying a number of histories in rheumatic children one finds that the rheumatic taint is often brought out apparently in a series of isolated events

such as arthritis, tonsillitis, chorea, erythema nodosum and pleurisy occurring at varying intervals and spread out over the greater part of childhood and adolescence. If however, a close watch is kept on such cases slight evidence of the activity of the rheumatic process in the form of rheumatic pains and stiffness in muscles and joints is often forthcoming, even in the apparently quiescent periods. Unless, therefore, it is checked at its commencement the disease must be looked upon as a continuous process with periodic exacerbations which may occur in many forms.

How are we to explain the occurrence of relapses and recurrences which sometimes are a feature even after treatment? This question has not yet been satisfactorily answered, but it is probable that one or more of several factors operate in determining these. The possibility that the treatment has not been thoroughly carried out at once suggests itself and in that case a certain residue of infection lurking somewhere in the tissues, e.g. in the tonsils or the periarticular tissues of the joints is probably responsible. If, as [15]Poynton & Payne believe, the streptococcus rheumaticus is a normal inhabitant of some throats, and if the conditions which favoured

the onset of the first attack persist, then repeated invasions by this germ seem likely unless they can be prevented. Lastly, there remains the possibility of a reinfection from outside the body, and this factor probably accounts for those cases which, many years after a complete recovery from a first attack, have a recurrence.

In connection with the last two views we must remember that the bulk of rheumatic patients must perforce remain in the unfavourable environment in which they contracted the disease.

The following cases show typical histories of rheumatism:-

D.K. aged 14 - female. At 12 years she had an attack of arthritis with endocarditis. Almost immediately following this she had chorea. After that she had an apparently quiescent period of about 18 months, but during that time although she had no definite arthritis, she frequently suffered from slight muscular pains in the back and legs for which she did not seek medical advice. At 14 years, her present age, she was again confined to bed with arthritis and slight pyrexia and has now a well marked mitral incompetence.

J.C. aged 10 - male. On December 6/23 he came to me complaining of frontal headache, stiff neck

and pleurodynia. The temperature was normal, pulse rate 90 per min. and there was no dilatation or alteration in the heart sounds. These symptoms cleared up in two or three days with rest and salicylates. He was kept in bed for 10 days.

On December 19/23 he had a relapse with stiff neck and pain in the upper abdomen. Temperature 100°F. Heart apparently unaffected. He was kept in bed for 3 weeks and required treatment for anaemia afterwards.

On February 7/24 he had a slight arthritis in the left hip with no obvious swelling. The heart was still apparently unaffected, but he was kept in bed for a month.

On April 21/24 he was sent home from school because the teacher had noticed that he was nervous and fidgety. He had no definite choreic movements and went back to school after a rest at home for two weeks.

On June 30/24 I was called to see him for a slight arthritis of the left hip, pleurodynia and pharyngitis. Temperature normal. No cardiac dilatation or murmurs. He had another rest in bed for three weeks. Since then he has had no further attacks.

M.T. aged 22 - female. At 10 years she was confined to bed with an attack of arthritis and endocarditis. At 13 years she had another attack of arthritis. Minor attacks of pain and stiffness in the joints for which she did not seek advice between 13 and 17 years. At 18 years she was again confined to bed with arthritis, endocarditis and pericarditis. Since then she has seldom been quite free from stiffness and pains in the joints, and has had several attacks of muscular rheumatism in the form of stiff neck and pleurodynia. Recently at the age of 22 years rheumatic nodules have appeared on one elbow, and she has now a double aortic and a mitral systolic murmur.

Cardiac Manifestations.

In children the three types of cardiac lesion due to rheumatism viz. endocarditis, pericarditis and myocarditis often occur together, but usually only either in very severe attacks or in cases where repeated infection has severely damaged the heart and a fatal termination is in sight. Then pericarditis and myocarditis are superimposed on a pre-existing endocarditis. This general carditis is often associated with rheumatic nodules the presence of which has a serious significance.

Endocarditis. In contrast to pericarditis is usually an early cardiac manifestation. It is probably always associated with some degree of myocarditis. In a fair proportion of cases it develops during an acute attack of arthritis, when there is very little likelihood of its being missed.

A pulse which is rapid and sometimes irregular and a rise in the temperature or a persistence of fever after arthritis has subsided may rouse suspicion. On examination of the heart we may find the apex beating well outside the mid-clavicular line, the cardiac dullness increased to the left and a blowing systolic murmur in the mitral area with accentuated pulmonary second sound. These physical signs may indicate the presence of an endocarditis but on the other hand they are sometimes due to myocarditis alone. It is often impossible to say immediately whether an endocarditis is present or not. Fortunately immediate diagnosis between the two conditions is of no practical importance as the treatment in the acute stage is the same in both cases.

If the systolic murmur changes its character from day to day - becomes louder and is propagated well into the axilla and to the back or takes on a musical quality - we are probably dealing with an acute endocarditis. The appearance of an aortic

diastolic murmur would clinch the diagnosis.

Unfortunately in children endocarditis more commonly develops insidiously and is frequently discovered accidentally during an examination.

Many of these cases on close enquiry are found to have suffered from 'growing pains' or a slight arthritis of one or more joints some time previously.

Such slight arthritic or muscular pains, indeed frequently direct attention to the heart where a well marked endocardial lesion may be discovered. In other cases there is no history of pain at all, and the endocarditis occurring primarily may develop undetected until attention is drawn to the heart by an attack of chorea, arthritis or tonsillitis.

It is quite a common occurrence on being called in to a case of rheumatism to find a slight arthritis affecting two or three joints and on examining the heart an acute endocarditis already present. In a fair number of cases the doctor is called in merely because the child is feverish and has been listless and off its food for some days or a week or two.

On closer enquiry the mother may remember that the child had complained of soreness in one or more limbs in a vague way or may remember that one joint - often the knee joint - looked slightly swollen recently. On examination the temperature may be anything from 100°F. to 103°F. with a rapid pulse,

and a mitral systolic murmur is present.

Such cases are sometimes brought into the consulting room by an anxious mother who has noticed that the child has been listless and disinclined for play lately and inclined to get out of breath on slight exertion.

A history of growing pains may be forthcoming and on examination a well established mitral systolic murmur is often found.

The temperature may be normal or slightly raised and the pulse rate is increased.

The following cases illustrate the insidiousness of the attack on the heart:-

D.H. female, aged 7, was brought to see me by her mother because she had shown slight stiffness in her right leg on walking and was complaining of pain in her ankles. The mother also volunteered the information that the child had been very 'nervous and fidgety lately'. On questioning her she remembered that for some considerable time before the child had suffered from 'growing pains' at intervals. On examination there was a little tenderness of the ankle joints but no obvious swelling. A loud harsh mitral systolic murmur propagated well into the axilla and to the back was present, and the left border of the heart was well outside the mid-

clavicular line. There were definite choreic movements in both arms and slighter movements of the legs and head. Temperature was 100°F. and the pulse 120 and irregular.

The chorea cleared up in about three months but although dilatation rapidly diminished the mitral murmur persisted in spite of prolonged rest in bed and salicylate therapy.

M.B. female, aged 4. I was called in to see this child for feverishness and pains in her legs. For a week or two before she had frequently complained of being tired, was listless and showed disinclination for playing and running about.

When I saw her she looked ill with dark rings round the eyes. Temperature was 101°F. There was slight swelling and a little tenderness of both ankles and knees. A loud musical systolic murmur was present in the mitral area and an accentuated second sound in the pulmonary area. The heart was slightly dilated. The temperature and arthritis subsided in a few days with administration of salicylates, but although she was kept in bed for two months the mitral systolic murmur persisted.

Systolic murmurs discovered during an attack of acute rheumatism vary considerably in their significance.

Soft blowing murmurs described as 'haemic' best heard at the base of the heart especially over or just external to the pulmonary area are frequently present especially if there is considerable anaemia. These are purely functional and disappear with improvement in the patients general condition.

Systolic murmurs at the apex may be either functional or organic. A functional murmur of relative incompetence of the mitral valve due to dilatation of the left ventricle occurs in some cases of acute myocarditis. This murmur as already mentioned is difficult to distinguish from that due to endocarditis.

It is soft and blowing in character, is usually more localised and frequently lacks any selective propagation backwards. This murmur, like the haemic murmur, is not in itself of any serious significance. It usually disappears completely with treatment. Harshness and a musical quality of a mitral systolic murmur may be looked upon as evidence of its organic nature. At the commencement of an endocarditis, however, the murmur may be soft and blowing. If after the subsidence of fever the increased rate of the heart persists, evidence in favour of valvular damage is strengthened. If on the other hand the rate becomes perfectly normal, and especially if

sinus irregularity appears, the heart may be considered to have escaped infection in spite of the presence of a mitral systolic murmur.

Whether the endocarditis causing a mitral systolic murmur is recent, old or recurrent has to be decided largely from the history. If it is old there will probably be a history of attacks of arthritis, endocarditis or chorea, dyspnoea, etc.

The organic systolic murmur is caused by a mitral leakage from the left ventricle to the left auricle due to thickening and rigidity of the cusps and chordae resulting in imperfect closure of the valve. This is by far the most common valvular lesion in acute rheumatism. The murmur is usually associated with an accentuated pulmonary second sound.

Once endocarditis is established the systolic murmur is permanent although it may vary in intensity from time to time. Following the first attack the lesion may progress insidiously, but often repeated subacute attacks of arthritis and muscular rheumatism of varying severity show that the rheumatic process is still alive in the patient, and with each attack, the endocardial lesion tends to become worse.

However, in estimating the value to be attached to an organic mitral systolic murmur, it is essential

also to consider to what extent the myocardium has become implicated, as it is on the efficiency of the myocardium rather than on that of the valve that the patient's future expectation of life must be based. According to the modern view it is doubtful whether mitral incompetence in itself ever causes serious embarrassment to the heart. The patient's response to effort, the presence or absence of abnormalities in rate and rhythm or increase in the size of the heart must all be considered in conjunction with the murmur.

Diastolic Murmurs heard over the heart in rheumatism are produced either at the aortic or the mitral valve. They are always due to organic lesions of the valves.

An aortic diastolic murmur may appear during an acute attack and then it is usually accompanied by a mitral systolic murmur, but more often the aortic murmur appears at a later stage. It seldom occurs by itself. [16]Poynton & Payne believe that the lesion is more common in the young than is generally realised. I have at present three girls under my care ranging between the ages of 16 and 22 all having an aortic diastolic and a mitral systolic murmur. All of them are advanced cases who have had recurrent attacks of rheumatism for the earlier part of which I did not attend them.

The murmur is heard best over the aortic area and is propagated down the left half of the sternum, but occasionally it is also heard well near the apex. It is due to backward leakage through the mis-shapen aortic cusps.

In some cases the diastolic murmur is associated with an aortic systolic murmur heard best over the aortic area and propagated upwards along the large vessels into the neck. This murmur is due to roughening and deformity of the valves. It is however, the diastolic element in the double murmur which is important as it always indicates a dangerous lesion.

Sometimes the systolic murmur of mitral incompetence is associated with a reduplicated second sound in the mitral area which according to [17] Cheadle is probably due to thickening and rigidity of the cusps. In time as the disease progresses the later of the two second sounds may be replaced by a soft blowing diastolic murmur. Later the diastolic murmur may immediately follow the two second sounds and eventually this may develop into a definite harsh presystolic murmur. These murmurs are best heard at the apex or just internal to ~~is~~ *it*. They represent phases in a gradual stenosing of the mitral valve

and are produced by the blood flowing from the left auricle into the left ventricle through the stenosed valve.

In favourable cases if they are treated by prolonged rest before the last stage is reached the murmur may retrace its steps until finally it disappears altogether.

In some cases accentuation of the first mitral sound is the earliest warning of the stenosing process.

The development of the presystolic murmur is essentially an insidious process and takes months or years. For this reason it is not found in an acute attack of rheumatic fever, unless there have been previous attacks, and for the same reason also it is rare in young children and becomes increasingly common after puberty. When it makes its appearance in early life it is of grave prognostic significance as it indicates that the stenosis is progressing at a comparatively rapid rate.

In these cases also, there is usually considerable myocardial damage which plays an important part in eventually bringing about fatal heart failure. It may involve the A.V. bundle causing partial heart block. In the later stages of mitral stenosis it

frequently causes auricular fibrillation. When this condition sets in the presystolic murmur disappears and only the apical diastolic murmur is heard.

I have been struck by the large number of adults examined for other ailments in the routine of general practice who have definite presystolic murmurs which have never been suspected, and which have apparently given rise to no symptoms. Others who have reached middle age or over, seek advice for the first time for early symptoms of cardiac failure. In both these classes of cases there is often no history of rheumatic fever and the patient cannot recollect ever suffering from any of the other ailments which we associate with the rheumatism of childhood. Indeed these cases are so common that one is confronted with the question as to whether, in the absence of a rheumatic history, they can all be considered to be due to acute rheumatism.

To answer this question satisfactorily it would be necessary to watch a large number of cases who have shown any, even the slightest, of rheumatic manifestations from their first appearance for a period of many years throughout childhood, adolescence and early adult life.

Another possible explanation of the frequency of this murmur is that it may not alway be due to

stenosis of the mitral valve. [18]Voelcker points out that presystolic murmurs are sometimes heard where there is actual dilatation of the mitral orifice as seen post-mortem.

The Temperature in the endocarditis of children is seldom high - usually ranging between normal and about 104°F. The higher temperatures from 102°F. to 104°F. usually occur only in the more severe attacks when there is considerable arthritis or severe tonsillitis or when the endocarditis is only a part of a severe pan-carditis. Hyperpyrexia is practically unknown in the rheumatism of children.

The Pulse rate is often increased out of all proportion to the rise in temperature being dependent largely on the extent of associated myocardial involvement. Irregularity of the pulse with an increase in rate is usually due to myocarditis. A persistently rapid pulse after fever has subsided may indicate that infection of the heart is still active, but on the other hand it may be due to loss of tone of the cardiac muscle and anaemia. If the appearance of a marked sinus irregularity with a return to the normal pulse rate is now generally considered to be a sign than the heart has escaped infection. In the final stages of mitral stenosis

the completely irregular low pressure pulse of auricular fibrillation is common.

Cyanosis and dyspnoea may be considerable in severe cases of pan-carditis and in chronic advanced cases, but these symptoms are as a rule less marked in children than in adults.

Myocarditis. This lesion is probably present to a certain extent in all cases in which the heart is affected in rheumatism. It may be associated with endocarditis, in which case it will be partly responsible for the dilatation present, or with pericarditis or with both.

In a fair number of acute first attacks in which a mitral systolic murmur is present the murmur disappears quickly with rest and salicylate therapy. In these cases the heart has probably escaped an endocarditis the murmur being due to the dilatation accompanying a myocarditis. In such cases the heart may return to the normal in every way as illustrated by the following case:-

R.P. Male, aged 11. I was called in to see this boy because of pain and swelling in his ankles and knees. His temperature was 101° and pulse 110. On examination a blowing mitral systolic murmur propagated well into the axilla was found. The left border of the heart was half an inch outside the mid-clavicular line. He was kept in bed for six

weeks and sodium salicylate was given in large doses.

The dilatation rapidly diminished, the murmur gradually became less marked and had disappeared altogether about two weeks before he was allowed to get up. At the end of six weeks his pulse rate was 86 with sinus irregularity and the apex was inside the midclavicular line. Six months later I examined him and found him in good health. The pulse was normal and there was no murmur or dilatation.

There had been no rheumatic symptoms of any kind in the interval.

In a certain proportion of cases there is no mitral murmur and dilatation of the heart with rapid pulse and excitable action are the only features of myocarditis.

In the progressive cases of arthritis in which there are repeated subacute attacks of endocarditis or pericarditis, the heart muscle suffers with the endocardium and pericardium and a chronic myocarditis is developed. This, in the chronic valvular disease of later life, is quite as important a factor in the production of ultimate heart failure as is the valvular lesion itself - if indeed, it is not more important than that lesion.

In the heart disease of children, death, if it occurs is due not to mechanical failure of the valves of the heart but to repeated reinfection of

the myocardium causing degeneration of the muscle fibres and interstitial fibrosis; thus the clinical picture of heart failure with dropsy and marked cyanosus as seen in the adult is seldom seen in children unless adherent pericardium has developed. Pericarditis is much less common in children than endocarditis, but, when it does occur, it is more immediately dangerous. It is the chief cause of death in the rheumatism of childhood. It may occur as a first symptom of rheumatism, but more usually it is associated with endocarditis and arthritis, and may occur together with any of the other rheumatic manifestations. When associated with endocarditis it usually develops when the endocarditis has become established. Rarely pericarditis is a feature of a rapidly developing carditis occurring in an acute attack of rheumatic fever with rapid effusion into the pericardial sac, high temperature and vomiting; leading ultimately to a fatal termination. Much more commonly it is subacute, the first attack being more or less recovered from only to be followed at intervals by further attacks. The patient gradually goes downhill. Endocarditis and myocarditis develop side by side with the pericarditis, anaemia becomes progressively worse and the child dies in the course of a few months or years from exhaustion or cardiac failure

without dropsy.

When pericarditis develops the child is usually already in bed owing to an arthritis. Endocarditis is usually present. One day the child is obviously worse, lies very still in bed with an anxious look and often a drawn expression, about the lower part of the face. The face is very pale and the lips cyanosed. He is dyspnoeic and may complain of praecordial pain if old enough. A short suppressed cough is common. The pulse is rapid, soft and tumultuous. The temperature is variable. It may be very slightly raised 99 - 100°F. On the other hand I have seen it go up to 104°F. in cases which have recovered. There is diffuse pulsation over the praecordium. A pericardial thrill is usually felt on palpation and a friction murmur, creaking in quality, heard over the base or over the whole heart. The murmur may be systolic in time or may be a double rub occupying a part of systole and diastole but not corresponding accurately to the onset of either. Both thrill and murmur, however, may be entirely absent and yet a pericarditis may be present. In that case, in the absence of obvious pericardial effusion the diagnosis is often difficult but should rest on the symptoms and on the condition

of the pulse. There is usually an associated mitral systolic murmur which may become louder as the pericarditis develops, or, if not already present, a murmur may arise.

Dilatation due to an associated myocarditis is often considerable and may vary from day to day. Its degree and rate of development are important guides in prognosis. An acute effusion into the pericardial sac is rare in children, and, if it does occur, is seldom serious enough to require tapping. As a rule friction, if present, persists for a few weeks and then gradually disappears as the case improves. It may reappear later with subsequent attacks, but very often the fibrosis progresses steadily in the absence of friction, while clinically there is increasing cardiac distress with dilatation and hypertrophy of the heart. In cases surviving long enough adherent pericardium eventually develops with the clinical picture of mitral disease with dyspnoea, cyanosus and dropsy as seen in adults and special signs of adherent pericardium.

Adherent pericardium is the end result of the chronic relapsing or progressive pericarditis, when the patient does not die at an earlier stage from exhaustion and failure of the heart muscle. The clinical features of heart failure in these cases

may be due either to an indurative mediastino-pericarditis, or to the formation of a firm fibrous sac often of considerable thickness shutting in the heart with complete adhesion between the two layers of the pericardium.

In the former case the heart fails because it is seriously hampered in its action by the adhesions to the chest wall and diaphragm. There is progressive dilatation and hypertrophy, dyspnoea, cyanosis and dropsy. Pulmonary and hepatic congestion develop, and are sometimes intensified by pressure on the large veins and pulmonary arteries in the mediastinum by masses of fibrous tissue. Clinically the adhesion to the outside structures is demonstrated by a fixed apex beat and Broadbent's sign.

In the latter case the circulatory failure is due in the first place to interference with the proper contraction and dilatation of the heart by the inelastic fibrous pericardial sac and in the second place to the inability of the enclosed heart muscle to develop in proportion to the progressive development of the rest of the body during childhood.

The following two cases recently under my care illustrate most of the features mentioned above:-

M.C. aged 4 years - female was first seen after she had been complaining of vague pains in her legs

for a week or two. A slight arthritis was then found affecting both knees and ankles. Temperature was 101°F. and pulse 120, and there was a loud mitral systolic murmur propagated into the axilla. There was slight dilatation.

The temperature and arthritis subsided in a few days under salicylates and rest. Dilatation disappeared and the murmur became less marked.

At the end of six weeks except for a persistent mitral murmur there was no sign of infection. The pulse was 90 and regular. The temperature had been absolutely steady for several weeks. Then just as I was considering the advisability of getting the child up I was called in one night because she had had a relapse. I found her in a serious condition lying quite still and helpless. Her face was deadly pale with dark rings round the eyes and cyanosed lips. She was breathing rapidly with ali nasi working. There was a short irritable cough and she complained of pain over the heart. The pulse was soft and feeble and irregular and the rate 150 per min. Temperature 104°F. Diffuse pulsation was visible in the praecordium and the apex beat was found about an inch outside the midclavicular line. A well marked pericardial thrill was felt and pericardial friction heard all over the heart in addition

to the mitral systolic murmur which had become louder.

The child was in a critical condition for two weeks the temperature remaining high for more than a week in spite of large doses of salicylates.

During this time she had two bouts of vomiting. Insomnia was troublesome. Then her condition again gradually improved, friction becoming less marked and finally disappearing altogether.

Dilatation at the same time gradually diminished although a loud systolic mitral murmur persisted.

At the end of three months she was allowed to get up with a slow regular pulse and no symptoms.

She was soon running about and playing without showing any signs of distress. About a month later she was brought to me with erythema nodosum on the anterior aspects of both legs and slight pains in the legs, but no definite arthritis. There was no pyrexia and the pulse slightly increased.

There was no friction over the heart. A further rest in bed for about two weeks followed and the erythema and pains soon disappeared. Shortly afterwards she emigrated with her parents to Australia. In a recent letter to her relatives in this country her mother wrote to say that the child had not had any recurrence of symptoms during the nine months after emigration and was playing about quite actively with other children.

Probably the change to a better climate and more suitable surroundings than those of a Lancashire town is the best thing that could have happened to her.

H.H. aged 10 years - male. On Nov. 11/21 I was called in to see this boy who was complaining of a "sore throat", pain in the legs and ankles and a slight cough. A slight arthritis was found affecting both ankles and knees. There was a loud mitral systoloc murmur and a diffuse general pharyngitis. Temperature was 102·2°F. and the pulse 110. He was at once put to bed and large doses of sodium salicylate given. The following day pain had disappeared from the joints and there was little swelling but the temperature remained the same. He now complained of a stabbing pain in the left side and the cough was short and suppressed. Slight pleuritic friction was found on the left side outside the mid-clavicular line and there were a few crepitations at the right base. On Nov. 13 the temperature had dropped to 101°F. The joints had cleared up but the chest condition was unchanged. On Nov. 14 I found that he had had a sleepless night, the cough was worse, the pain less severe and there was white frothy expectoration streaked with blood. He was dyspnoeic and his lips were cyanosed.

Temperature 103°F. Pulse 120. There were definite broncho-pneumonic patches in both lungs and pericardial friction was heard all over the heart, the left border of which was well outside the midclavicular line. From this time onwards the temperature ran an irregular course fluctuating between 99°F. and 104°F. until December 3 when it reached normal. On Nov. 17 there was deafness in both ears without pain or discharge lasting only one day. The pharyngitis cleared up in a few days, but the broncho-pneumonia persisted until the fall of the temperature on December 3. and then quickly cleared up, all physical signs having disappeared by December 9. Pericardial friction continued to be marked till about December 3 and then gradually became less until its complete disappearance by December 12. The mitral systolic murmur and some dilatation persisted. The pulse did not fall with the temperature but continued to be rapid and excitable.

On December 16 there was a relapse with pain in the fingers of the left hand, and slight pericardial friction returned near the apex. The temperature rose to 100°F. and the pulse to 120. Friction disappeared in two days and the temperature was normal in three days but shifting pains in the hands, wrists, left should and knees continued until December 21.

After this with the exception of one rise to 101°F. on December 30 the temperature remained normal for a little over a month and in this interval no symptoms were complained of but the pulse rate continued at about 100 per minute.

On Feb. 17/22 he complained of praecordial pain and breathlessness, but there was no pericardial friction. Temperature 101°F. Pulse 130. The pain disappeared and the temperature dropped to normal in a few days but he had a considerable degree of anaemia by this time, the heart was much dilated and the pulse rate continued at about 120. In spite of large doses of Salicylates, Digitalis and Parrish's Food together with rest, his general condition bad not improved much by March 25.

I then lost sight of him until July 21 when he was brought to me suffering from a conjunctivitis of the right eye. There was a loud mitral systolic murmur and diffuse pulsation over the praecordium with systolic indrawing at the apex which was well outside the midclavicular line. The temperature was normal and the pulse rate 90 per minute. The conjunctivitis cleared up in a few days.

I did not see him again until Nov. 22/23 when I was called in after he had been suffering from pains in his joints and pain over the heart for three weeks.

There was no swelling of any of the joints. The temperature was normal and the pulse rate 108. Slight pericardial friction could be heard on December 3 but this soon disappeared.

I never saw the child again but heard later that he had died in October 1924, i.e. three years after the commencement of the first attack.

This case is interesting not only because of the pericarditis, but because it gives us a picture of nearly all the manifestations of the rheumatism of childhood occurring in one patient in a comparatively short space of time in contrast to the more usual slow procession of events.

Thus we have an arthritis, tonsillitis, pleurisy, broncho-pneumonia, endocarditis, pericarditis and myocarditis all developing within a week. Unfortunately there were long intervals during which the patient was not under observation. This cannot always be avoided even in general practice. Otherwise a more complete picture would have been seen. Arthritis in the rheumatism of children tends to be mild especially in younger children from 4 to 7 years of age in whom it is extremely uncommon to see anything like the severe rheumatic arthritis of later life.

Frequently there is complaint only of a little pain in one or more joints with very slight swelling made out only on close examination, or no swelling at all. Usually there is a little tenderness on pressure.

In this mild arthritis there is seldom a temperature above 100°F. except in cases where there is an associated endocarditis, pericarditis or tonsillitis in which case a temperature of 102 - 104°F. may be reached. These cases are often missed because they are looked upon as feverish colds or labelled influenza by the parents. The child is kept in doors for a few days till the symptoms pass off and no more is thought about it, but an endocarditis frequently develops during such an attack. This no doubt accounts for many of the cases of valvular disease in children discovered accidentally during an examination for some other ailment or in the course of school medical examinations. During a short period while deputising for a school medical officer I came across several such cases in which there was no history of rheumatism.

In rather older children from seven upwards it is not uncommon to find a more severe arthritis, although this seldom reaches the severity of an acute arthritis as seen in adults.

The child complains of severe pain in one or more joints most commonly the ankles, knees or wrists, less commonly the elbow, hip or shoulder joints. He lies very still in bed in a helpless attitude as any movement increases the pain. He cannot bear the affected joints to be touched because of their exquisite tenderness. The joints are obviously swollen. They are held in the position of greatest ease; thus when the ankle joints are affected the toes often drop, and when the child is asked to dorsi-flex the ankle he is apparently unable to do so. In one case under my care this dropping of the foot led me to suspect acute anterior poliomyelitis at first sight.

I was called in one evening to see a child of 6 years because he had suddenly become 'paralysed' in one leg. The child was feverish and complained of headache and pain in one leg. There was no obvious swelling of any of the joints, but both the ankle and knee joints on one side were tender. Nothing would induce the child to dorsiflex the foot on the affected side or move the limb at all.

On examining the chest I found a loud mitral systolic murmur which removed any doubt as to the diagnosis.

The joints are often involved symmetrically.

Thus when the case is first seen there may be arthritis of both ankles, both knees or both ankles and knees. Pain is usually actually complained of only in the more severely affected joints. Such pain often passes off in a day under treatment and the next day the pain may be complained of in another joint or in another set of joints which, although affected before, had given rise to comparatively little discomfort. Thus the impression is given that the arthritis flits from joint to joint whereas it is merely a question of the relative severity of the affection in different joints.

The swelling disappears more slowly than the pain being often noticeable two or three days after the pain has subsided or been considerably relieved. Pain is sometimes referred away from the joint which gives rise to it. A common example of this is the pain of arthritis of the hip joint which is often referred to the iliac region, simulating appendicitis when occurring on the right side.

In the majority of cases arthritis subsides in a few days, but in a certain proportion of cases a degree of stiffness is left which is very difficult to get rid of. For this the periarticular tissues are responsible rather than the articulation itself.

It occurs especially when effective treatment has not been carried out from the commencement in a first attack. From time to time these joints become more or less painful and the stiffness is increased, while acute exacerbations may occur superimposed on the subacute process, which is often associated with fibrositis in other parts of the body and with progressive endocardial or pericardial lesions.

Fibrositis. In children rheumatic pain frequently arises from an affection of the muscles, tendons and fasciae rather than the joints, a fact which is not sufficiently realised - sometimes with disastrous results for the patient. This is especially true of the milder attacks in young children and of the subacute rheumatism of progressive cases.

The muscles, tendons, fasciae and bursae in the neighbourhood of joints may be affected concurrently with an attack of acute arthritis, but very often they are the only structures affected. Thus the tendons of the ham strings are not uncommonly affected alone. The child complains of pain and walks on the balls of his toes with the knee bent. The mother brings the child for advice because she thinks he has sprained his ankle or hurt himself in some way by a fall. There is usually no temperature. A few days of rest in bed and salicylates will clear

up the condition. In one case under my care the adductor muscles of the thighs were repeatedly affected. A definite attack of arthritis eventually developed some months later involving both knees and one hip joint.

Pain in the chest arising from fibrositis of the intercostal or pectoral muscles, because of its stabbing character and aggravation by deep breathing leads to a suspicion of pleurisy but no friction can be elicited. A rheumatic fibrositis of the muscles and fibrous tissues of the neck not uncommonly causes stiff neck and in some cases even torticollis. This is especially apt to occur in progressive cases associated with stiffness and pain in the joints. The readiness with which it yields to salicylates together with its association with other rheumatic manifestations is proof of its rheumatic origin.

Epigastric pain occurring suddenly without vomiting or other gastric disturbance in the absence of pleurisy, pericarditis or endocarditis and with very little tenderness has been described by a few writers as an early sign of rheumatism.

It is probably due to a fibrositis of the lower intercostal muscles or the abdominal muscles. I have seen it in one case only in a child who was complaining of 'growing pains' but so far no more serious manifestation has arisen.

Muscular wasting is a common feature of severe rheumatism and is especially marked after a bad attack of chorea.

Growing Pains. The nature of these is disputed, some people believing that they have nothing to do with rheumatism. There is no doubt, however, that they occur in a large number of children who subsequently develop unmistakable rheumatic lesions. In most of the cases which I have had the good fortune to observe from the commencement 'growing pains' have been among the early symptoms. I consider it a safe rule to look upon these as an early warning of acute rheumatism and to treat them accordingly. They are usually vague pains in the legs or less often in the arms, but sometimes they are so severe as to make the child cry. There is no associated tenderness or swelling. They are seldom mentioned spontaneously by the mother, and a history of growing pains is only elicited on close enquiry when the child has been brought in with a 'sore throat', a 'feverish cold' or one of the other manifestations of rheumatism.

Tonsillitis. It is impossible to say with certainty whether a particular attack of tonsillitis or general pharyngitis occurring by itself is rheumatic in origin,

because there is no certain distinguishing feature between a rheumatic 'sore throat' and those due to many other causes. The best policy is to regard all such attacks occurring in children as rheumatic unless clinically or bacteriologically they are proved to be due to some other cause.

In most cases in which there is tonsillitis it is an early manifestation. It may precede a rheumatic arthritis by a few days or may occur simultaneously with it. In either case the condition usually clears up in a few days.

Tonsillitis, endocarditis and arthritis commonly occur together. In my own cases tonsillitis has not been a prominent feature. Many children go through several attacks of rheumatism without any perceptible change in the throat.

This raises some doubt as to whether the throat is always the portal of entrance of the rheumatic infection, unless, of course, the organism may find its way through the mucous membrane of the throat or through the tonsils without causing any obvious change.

The inflammation is usually not confined to the tonsils but involves the pharynx generally. This probably accounts to a certain extent for the fact that the removal of the infected tonsils does not always prevent recurrences.

Nervous Manifestations.

Of these chorea is by far the most important, but many children are nervous without ever developing chorea. The rheumatic child is liable to headaches, is often highly strung, excitable and intelligent at school. In a proportion of cases this nervousness develops into a pre-choreic state. The child becomes fidgety and unable to concentrate his attention on his work, his hand writing deteriorates and he tends to drop things. He may be reprimanded or punished for his inattentiveness or carelessness and such punishment is sometimes the exciting cause of a typical choreic attack.

Chorea. The rheumatic origin of chorea is still disputed. Pathologically [19]Poynton and Payne have demonstrated the diplococcus rheumaticus in the brain and meninges of cases of chorea. Clinically we know that chorea frequently comes on during an attack of rheumatic arthritis. It may also be associated with any of the other manifestations of rheumatism and especially with endocarditis. It may occur at any stage of the rheumatic history of a child. It may be the first symptoms of the disease. In all cases of chorea that I have attended there has been either a previous rheumatic history, an associated rheumatic lesion or a subsequent attack of rheumatism in one of its many forms.

The association is so close that to me it seems impossible to doubt that chorea is a truly rheumatic manifestation.

Poynton and Payne give the following remarkable figures in support of the rheumatic origin of chorea:- Out of 217 cases 122 gave evidence of heart disease and other rheumatic manifestations. In 28 more there were arthritis and muscular pains. In another 22 cardiac dilatation. 10, again, followed a "sore throat". 20 of the remainder gave no history of any cause, but two of them later developed acute rheumatism. 15 were attributed to fright and shock but were noted to be rheumatic in later life. 8 were attributed to strain at school.

The following cases which have been under my care show the close association of chorea with rheumatism.

T.C., male, aged 12. In March 1923 he had an attack of pharyngitis with severe frontal headache. In May 1924 he had an attack of chorea with arthritic pain in the knees but no swelling. In August 1924 he had conjunctivitis of the right eye. In September 1924 he had arthritis of the left ankle and knee joints with a temperature of 101°F, and endocarditis leaving him with a mitral systolic and a mid-diastolic murmur.

D.H., female, aged 19. At the age of 9 years she had arthritis and chorea which kept her in bed for several months. At 16 years she had a bad attack of "sore throat" although her tonsils had been removed early in childhood. At 19 years she developed another attack of chorea associated with muscular pains in the arms and legs and pain and stiffness with slight transient swelling in the wrists and ankles. There was no pyrexia. A slight presystolic murmur was present. The throat showed a general congestion while the remains of a tonsil on one side looked unhealthy with large open crypts.

E.G., male, aged 4. On October 15th 1924 he was brought to see me because his right leg was stiff and he could not stand on it. Both knees were slightly swollen and tender. There was no pyrexia and the heart was apparently unaffected. He was sent to bed. On November 5th 1924, while still in bed, he began to have choreic movements in the face arms and legs. The temperature remained normal and there were no cardiac murmurs. The movements gradually became worse and his mother had great difficulty in keeping him under the bed-clothes. A medical examination was a difficult procedure on account of the violence of his movements. On

On November 21st 1924 a mitral systolic murmur appeared for the first time and was associated with considerable dilatation of the heart. The temperature remained normal until December 3rd when there was a rise to 100°F, associated with headache, vomiting and diarrhoea. The temperature and gastro-intestinal symptoms subsided in two days. From this time onwards the symptoms gradually subsided. The murmur became less marked and on January 30th 1925, when the child was last examined he showed no sign of chorea, the cardiac dilatation had disappeared and with it the mitral systolic murmur.

Chorea is often associated with endocarditis, an unfortunate occurrence for the patient, because the violent movements of a severe chorea cannot but affect the damaged heart adversely. In many cases of chorea the heart is rapid and excitable without any murmurs. In other cases there is a soft systolic murmur due to dilatation which usually disappears altogether as the patient improves, but more commonly the systolic murmur is due to endocarditis and is permanent.

The slow progressive type of endocarditis which ends in a mitral stenosis appears to be more

common in rheumatic cases who have had chorea.

The condition may show itself merely as an exaggerated fidgetiness with perhaps slight choreic movements in one arm. On the other hand the movements may be so violent as to make it difficult to keep the child in bed. Between these extremes all degrees of severity occur. In moderately severe cases all the limbs as well as the head are usually involved, but the movements are usually more marked on one side. Some cases appear to be one sided, but on careful examination it is usually found that there are slight movements on the other side as well.

The movements are involuntary and purposeless. They are sudden and jerky. If told to perform a voluntary act the child does it quickly, because coordination is imperfect, and the act can only be performed successfully if done quickly. This is well illustrated by the quick grab the child makes for a pin when told to pick it up off the floor. Involuntary movements become worse when the child knows that he is being observed, and the presence of other children who tend to mimic is harmful, hence the value in treatment of isolation with some sensible adult.

Some cases show considerable muscular weakness and these constitute the "paretic type". Such children may suddenly fall down when walking, owing to a leg giving way. The paresis like the choreic movements may be more marked on one side than on the other.

The child is very emotional, laughs and cries with equal facility on the slightest provocation, but chorea insaniens is very uncommon in children.

In mild cases there is no pyrexia and even in severe cases the temperature usually does not exceed 100°F unless the chorea is complicated by endocarditis or arthritis, in which case it may be considerably higher. There is progressive anaemia and wasting in severe cases. The duration of the condition is anything from 10 or 12 weeks to 1 year. Recurrences are frequent. Few cases escape without a second attack.

Subcutaneous Nodules. In the evolution of the rheumatic nodule we have all the essential features of the tissue response to the invasion of the rheumatic virus. Clinically, however, it is one of the least common of the more serious manifestations of acute rheumatism.

Still[20] found nodules in 27.5% of 200 severe cases of rheumatism in children under 12 years of age, but only 10% in milder cases. Moon[21] found them in 10% of rheumatic cases.

While they are more common among younger children they do occasionally occur in older subjects. Two of my cases had passed the age of puberty before nodules developed. The majority of children who pass through repeated attacks of rheumatism never show them.

The significance of these nodules was first realized by Sir T. Barlow[22] and Dr. Warner, although they had previously been described by Hillier[22], Meynet and others. The former two observers considered them to be of serious import and subsequent clinical experience has confirmed this view. Their appearance, especially when in considerable numbers, is fraught with grave danger to the patient. Usually they are associated with endocardial, myocardial and pericardial lesions of a progressive type.

The nodules commonly appear in crops over bony prominences and along tendons in the vicinity of joints. The knee, elbow and ankle joints are the commonest situations, but they are also found

along the extensor tendons of the hands, especially over the knuckles, along the curved lines of the occiput and sometimes along the spinous processes of the vertebrae and the spines of the scapulae. They occur in numbers varying from 1 to 100 or more, and vary in size from a pin's head to an almond. They grow rapidly to reach their maximum size within a week. They are freely movable under the skin and at an early stage in their development continued pressure will make them less prominent. Even when scarcely visible they can usually be palpated. There is as a rule no tenderness but occasionally one or more of them are tender to the touch. The duration of a nodule varies from only a few days to several months, the longest period recorded being 5 months. I have now under my care a case in whom certain nodules which have been carefully watched, have lasted for 4 months and are as yet showing no signs of disappearing.

These nodules although truly rheumatic in origin are curiously resistant to salicylate treat--ment as illustrated in the following case:-

V.E., female was 17 years old when she first consulted me. She had Scarlet Fever at 6 years and her parents were then told at the Fever

Hospital that her heart was affcted. At 14 years she had a slight attack of pain and stiffness in her legs when at the seaside. When I was called in to see her for pains in her joints, I found a fading erythema nodosum over both tibiae and slight swelling and tenderness of both knees and elbows. Pulse was 120 per minute, temperature 100°F, and she was rather anaemic. A well marked mitral presystolic and a systolic murmur were present. Pain and swelling soon disappeared but stiffness in the knees and elbows persisted. She was kept in bed for seven weeks. Towards the end of this time several subcutaneous nodules were noticed on the occiput. These soon disappeared, but after she got up crops of nodules appeared on the knuckles of the right hand and along the extensor tendons of the fingers.

These were as large as a pea and not tender. They were made prominent when the skin was stretched over the knuckles by flexing the fingers. Salicy--lates did not seem to have any effect on them. Some disappeared but others arose in their place in different situations while the patient was taking large doses of sodium salicylate regularly. She was not very tolerant to that drug and a change

had to be made to Aspirin, but the nodules remained unaffected. A month later two large nodules, one of which was slightly painful and tender to the touch, developed on one elbow. In the meantime there was persistent stiffness in the knees and elbows, associated occasionally with pain but no swelling of the joints.

Attacks of stiff neck and lumbago also occurred. During exacerbations her pulse rate was increased but there was no rise in temperature. She complained of dyspnoea on walking and had attacks of palpitation.

Six months after I first saw her, nodules were still present. The cardiac murmurs had not undergone any perceptible change, but she still showed cardiac distress on exertion. After that I lost sight of her.

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SKIN MANIFESTATIONS.

Erythema Nodosum is in my experience the most common of these, but there is still much dispute as to whether the condition is purely rheumatic, part of an allied disease, or a manifestation of several different infections of which rheumatism is one. It is a difficult question to decide as one sees among children and adolescents so many cases of erythema nodosum unassociated with other rheumatic manifestations. That these may be only the first in a succession of rheumatic events is of course true, but I have not been able to trace the development of subsequent rheumatic attacks in such cases, although in some there have been family histories of rheumatism.

The condition does sometimes arise in undoubted cases of rheumatism but in my opinion the association is not common. It may occur during an acute attack of arthritis or in the subacute stage. In one of my cases who had an old established mitral lesion, an attack of erythema nodosum was just fading when a subacute attack of arthritis developed. Another child who had just recovered from an attack of arthritis with endocarditis and pericarditis, came into my consulting room with well marked erythema nodosum on both legs. On the other hand,

I have seen it in one case of early pulmonary tuberculosis with haemoptysis, and in another girl who had no sign of rheumatism or tuberculosis, but whose father died of pulmonary tuberculosis shortly after.

Erythema nodosum occurs chiefly on the legs, the favourite site being over the anterior surface of the tibia. Occasionally it is seen on the forearms confined chiefly to the extensor surface ~~of the tibia~~. The nodules may appear in crops with an interval of several days between individual outbreaks, but in cases which are treated there is usually only a single outbreak. At first bright pink in colour, the nodule passes successively through the stages of a bruise until it disappears. It is slightly raised above the surface, oblong in shape, about 1-1½" long and about 1" broad. It is usually exquisitely tender to the touch and gives rise to a smarting pain. Sometimes there is slight swelling and aching of a neighbouring joint. When the erythema occurs by itself there is usually no rise of temperature.

In spite of the uncertainty with regard to its pathology the occasional association of the condition with other rheumatic manifestations is

sufficient reason for regarding every case of erythema nodosum as a potential, if not an actual, heart case and treating it accordingly.

Purpura Rheumatica occurs chiefly in childhood. It may occur during an attack of rheumatic arthritis or may occur by itself in a rheumatic subject in which case the joints in the neighbourhood of the eruption are swollen and painful, although the temperature may be normal. The rash is practically confined to the limbs and is much more common on the legs than the arms. It may be the only skin affection present but often there are other skin lesions associated with it, such as urticaria or erythema nodosum.

Urticaria sometimes arises during an attack of rheumatic fever. It differs in no way from that due to many other causes. An attack developed in one of my cases three days after the onset of arthritis and endocarditis. The wheals were very large and the itching considerable. The rash had disappeared on the following day. As salicylates were continued in large doses after the rash had appeared, these could not be blamed for it.

Erythema may be scarlatiniform or blotchy, localised or generalised. In the diffuse cases it is often multiform and may then appear as a simple erythema on the face associated with marginate erythema on the limbs and sometimes a papular erythema chiefly on the knees and elbows. Considerable brown staining is sometimes left behind on the legs, especially over the tibiae, which is often first seen when the limbs are examined for an acute arthritis. As in urticaria one has to bear in mind that sodium salicylate may itself be responsible for the rash.

Psoriasis has been ascribed to rheumatism by some. I have never noticed any association between the two conditions, although psoriasis is comparatively common in general practice. Poynton and Payne[23] suggest that it may be the more chronic type of manifestation of the skin in rheumatism.

Anaemia. There is always more or less secondary anaemia after an attack of acute rheumatism in children. It is more marked after repeated attacks, when it may be associated with considerable wasting, and also after a severe attack of chorea. The anaemia is sometimes responsible for a persistently rapid pulse associated with soft

systolic murmur in all areas after prolonged rest in bed for an acute attack of rheumatism. The pulse rate is restored to normal and the murmurs disappear when the anaemia is treated.

RESPIRATORY MANIFESTATIONS.

Epistaxis occurs more frequently amongst rheumatic children than others and may be an early sign of rheumatism.

Pleurisy is common in rheumatic children. It is usually dry, and if pleurisy with effusion does occur tapping is seldom needed. It may occur at any stage. It is not infrequently associated with pericarditis.

Broncho-pneumonia occasionally occurs in association with a definite rheumatic attack. It differs in no way from broncho-pneumonia due to other causes. I have only seen one case. It is possible, however, that some of the broncho pneumonias occurring in children unassociated with other signs of rheumatism may be rheumatic in origin.

ABDOMINAL SYMPTOMS.

A rheumatic peritonitis usually confined to the upper part of the abdomen round the liver and spleen with loud peritoneal friction has been described, but the condition is very rare.

Digestive disturbances are not common in my experience. Vomiting does occasionally occur during an attack but is then usually due to the salicylates administered except in cases of tonsillitis and pericarditis in which vomiting apart from treatment is not uncommon. In the intervals between rheumatic attacks children sometimes suffer from diarrhoea.

Appendicitis. Nothing conclusive has been proved with regard to the relationship between appendicitis and rheumatism, and few medical men will at the present stage of our knowledge take the responsibility of treating a case of acute appendicitis without reference to the surgeon.

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TREATMENT.

Prophylaxis.

Until recently the spectacular effects of salicylate therapy in acute rheumatism seem to have lulled the medical profession into a false sense of security with regard to the treatment of that disease. Now it is being gradually realized that attention to the hitherto neglected aspect of treatment, viz. prevention, offers the best hope of reducing the incidence of cardiac rheumatism, and, with it, the number of cardiac cripples, at any rate, until such time as a specific antiserum possessing the effective--ness of Diphtheria antitoxin may be evolved, if ever.

Attempts at prophylactic inoculation have so far not met with great success. We are, therefore, obliged to turn our attention from a direct attack on the causal organism to eliminating these subsidiary factors in the etiology with which we are familiar. To accomplish this successfully it is necessary to attend in the first place to the proper instruction of the parents of rheumatic children. As the majority of cases of acute rheumatism occur among the children of the poor, it is upon the general practitioner, the lay health worker and Child Welfare Centres that this responsibility mainly falls. Parents should have explained to them the significance of growing pains, sore throats and painful swellings

on the limbs, and should be warned of the importance of undue nervousness, clumsiness and night terrors as premonitory symptoms of chorea.

They should have it pointed out to them that listlessness, breathlessness, anaemia and disinclination for play are sometimes the only symptoms of rheumatic infection.

Frequent re-examination of the rheumatic child, apart from acute attacks, is of the utmost importance and should be continued throughout child--hood and adolescence at intervals of about three months. To get the parents' consent presents little difficulty when it is explained to them that rheumatism is a disease which is likely to recur, and therefore to be constantly guarded against. Most parents have a great dread of "heart disease" and the mere mention of the possibility of this being the price of neglect on their part acts as a useful spur.

The question of suitable clothing requires attention. The child should wear woollen or silk undergarments, reaching from the neck to the ankles. The habit of clothing female children in a very short dress barely reaching half-way down the thighs, and very short stockings or none at all is a dangerous one in the case of rheumatic subjects.

Damp and chills should be specially guarded against particularly in spring and autumn when the danger is greatest. After a bath the child should be thoroughly dried. He should never be allowed to keep wet clothes or shoes on in school but should change immediately after a wetting.

With regard to diet, there is no reason why, apart from acute attacks, the child should not eat everything a normal child eats. Too much sweet stuff should be avoided. There seems to be no foundation for the popular belief that meat is bad for the rheumatic.

School medical examinations are very helpful in discovering a certain percentage of those cases of slight or insidious rheumatic infection which would otherwise be missed and allowed to progress without treatment.

Such cases should be sent to their private doctor for prompt treatment. It is also the duty of the School Medical Officer to instruct teachers regarding the danger signals of rheumatism in the child. While it is unnecessary to debar all children who have had a slight attack of rheumatism or have a family history of rheumatism, from active games at home or at school, over exertion both mentally and physically in the nervous type of rheumatism should not be allowed. If the child

becomes fidgety, inattentive and complains of headache, he should be sent from school to rest at home for two or three weeks. The same precaution applies to cases of slight sore throat.

Housing.

As the bulk of rheumatism is found amongst children living in the poorest houses, there is reason to hope that as the housing conditions of the poor are gradually improved we may look for a decline in the incidence of the disease. In choosing suitable dwellings for rheumatic subjects it is generally conceded that damp clay soils in river valleys and cold bleak hilly sites should be avoided. In the construction of houses the provision of proper damp-proof courses should be insisted on. Many of the older houses in the poorer quarters lack these and dampness is consequently rife. Multiple cases of Rheumatic Fever occurring in a house have lead authorities to believe in"infected houses", i.e. infected paste behind wall-papers.

Climate.

A change of climate is usually only possible for the few with ample means. For these a warm, dry climate should be advised.

In addition to educating the parents it falls to the lot of the general practitioner to deal

with the bulk of those early and trivial manifestat--ions of rheumatism which are still amenable to treatment. If these are most scrupulously dealt with in every case a great deal of more serious trouble will be prevented. Anaemia, nervousness, and listlessness should be treated by rest for two or three weeks away from school combined with some such tonic as Parrish's Food, cod liver oil, or iron and asenic.

Growing pains, stiff neck, pleurodynia and slight sore throat should be treated promptly by rest combined with salicylates in some form or other. Enlarged tonsils and adenoids should be removed and all carious teeth filled or removed. All obscure febrile conditions in children should likewise be treated by rest in bed and salicylates. It is gratifying to see the rapidity with which the bulk of such conditions clear up on the exhibition of sodium salicylate.

Methods of Treatment.

Sydenham[24] who first defined the disease treated it by blood-letting and diet. His teaching was followed until the middle of last century when blood-letting gave place to drug treatment in the shape of the almost equally drastic measure of purgation. Now although it is still considered

desirable to ensure regular action of the bowels, purgation in itself is not looked upon as a curative measure.

The Alkaline system of treatment was brought into prominence by Fuller[24] and was soon extensively employed in this country. It rested on the mistaken belief that rheumatism was due to an acid in the blood which it was hoped the alkalis would reduce with benefit to the patient.

Although as a treatment in itself it has largely given place to salicylate therapy alkalis are still advocated by most authorities as an aid to salicylates, and also to prevent the danger of acidosis and gastric irritation from the administrat--ion of large doses of salicylate.

Statistics compiled by the Selective Investigation Committee[24] support the contention of Dr. Fuller that the incidence of cardiac inflammation is reduced and its severity diminished by the administration of full doses of alkalis. This benefit is believed to be due to a depressant action on the heart which lessens the danger of the onset of endocarditis or pericarditis.

Salicylate Therapy and Willow Bark.

The use of willow bark in the treatment of fevers can be traced back more than 100 years. Salicin, its active principal, came into use for the

same purpose about 75 years ago. Thirty years later salicylic acid was introduced and first used mainly as an antiseptic, but soon after salicylic acid in Germany and salicin in this country were used in the treatment of rheumatic fever with such favourable results that the treatment by salicin, salicylic acid and its compounds soon became general. Salicylic acid being too irritable to the stomach has been entirely replaced by sodium salicylate in this country. Although it is generally admitted to be the most useful drug in the treatment of acute rheumatism, there exists a wide difference of opinion as to its mode of action and the indications for its use in the various manifestations.

According to one theory it acts by neutral--ising or destroying an infective toxin or toxins. In connection with this view [25] Syme suggests that one of the toxins of rheumatic fever may be a fatty substance which combines with, and is removed by sodium salicylate in the same way as that drug combines with the fatty acid, Glycocine, in the liver to form salicyluric acid which is then excreted in the urine.

[26] Miller, supporting the view that the drug has a bactericidal action in rheumatism, explains its failure to subdue such clinical manifestations as chorea and myocarditis on the grounds that these are

due chiefly to toxic parenchymatous changes, rather than to a direct invasion of the rheumatic organism, and that it cannot therefore be expected to do more than check the damage by eliminating the infection. In a similar way he maintains that rheumatic anaemia, nodules and large persistent effusions, being the results of an infection, may persist after that infection has been removed, while changes such as valvular deformities and adherent pericardium which are caused by healing processes cannot of course be altered by salicylates.

There are others who believe that the usefulness of salicin and the salicylates in rheumatism is confined to their non-specific action on pain and fever, and indeed, look upon this action as a source of danger to the patient in as much as it masks symptoms of serious conditions in the same way that morphia does in an acute abdominal condition.

It is in arthritis and pyrexia where the salicylates have their most remarkable effect. Pyrexia is considerably shortened, subsiding usually in about three days, if the drug is given in effective doses. Arthritic pain is quickly relieved and the joint effusion rapidly cleared up. Rheumatic tonsillitis, too, quickly responds to sodium salicylate. Unfortunately, these very definite and favourable results are not obtained in the cardiac manifestations. Here we find a very wide difference

of opinion as to the effect of the salicylates. [27] [27] Cheadle, Church and others advise caution in the administration of this group of drugs when the heart is found to be involved because of their depressant effect on the heart. Some even assert that the salicylates predispose to cardiac infection. [28] Syme, [29] Coombs, [30] Miller and others hold that the course of a cardiac inflammation is favourably influenced by them, especially when they are combined with an alkali. I have certainly never seen any bad effects on the heart which could be ascribed to them. It has been argued that the liability to relapses and recurrences supports the view that the salicylates have no specific action in rheumatism. In my opinion relapses would be less frequent if treatment with salicylates were more thorough, while recurrences are probably often due to reinfection from outside the body. [31] Ersthrom and Walberg in the proceedings of the Finnish Medical Society compared their hospital records of the 34 years preceding the introduction of the salicylates with those of the succeeding 44 years. They found that there was no diminution in the incidence of rheumatic endocarditis during the latter period and concluded that those drugs could not therefore be regarded as specific remedies. [32] Miller, on the other hand,

in support of his view that the incidence of acute pericarditis is falling, gives the following significant statistics from St. Bartholomew's Hospital Reports Vols. VIII - LII.

Acute Pericarditis per 100 Rheumatic In-patients.

1870 - 74	.....	13.57
1875 - 79	.....	10.96
1880 - 84	.....	12.07
1885 - 89	.....	9.18
1890 - 94	.....	13.18 (?)
1895 - 99	.....	10.90
1900 - 04	.....	9.53
1905 - 09	.....	4.88
1910 - 14	.....	5.09

In considering the question as to whether the salicylates prevent cardiac inflammation it is necessary that we should make allowance for the insidiousness of the attack on the heart. In many cases the heart is already affected when sodium salicylate treatment is begun. From my own experience I am convinced that if the drug is administered in effective doses before there is any sign of cardiac involvement, it does lessen the chances of cardiac inflammation by reducing the infectivity. But there is no doubt that the insidiousness of the attack on the heart is a great limitation to the success of sodium salicylate as a prophylactic against Carditis. For this reason, while making full use of the drug, it is necessary that we should pay more attention to other methods of prevention.

The anaemia of rheumatism, skin manifestat--ions and chorea are not obviously affected by salicylates. Miller's explanation for this failure seems to me a satisfactory one.

With regard to the dosage of sodium salicylate, it is generally agreed that the patient should be brought under the influence of the drug as rapidly as possible in acute attacks, and with this aim large doses are first given at short intervals and gradually reduced as the symptoms are relieved. In children up to 12 years of age, 6 to 15 grs accord--ing to age, and in older children 20 grs every two hours for about four doses, and then every four hours until pain has disappeared, will generally be found satisfactory. After that the same dose is continued three times a day for three weeks after all pain has ceased. Should it return during this period the frequency of the dose should again be increased, and, if necessary, a larger dose given.

There is much difference of opinion as to the dosage required for the manifestations other than rheumatic arthritis. Very large doses are given by some. Lees advocates as much as 600 grs in 24 hours in children. I have not found that larger doses of sodium salicylate have any more effect on the more resistant forms of rheumatism than the

ordinary dose which is effective in rheumatic arthritis with pyrexia.

Salicylism is sometimes produced if sodium salicylate is given in large doses for a long time. Children are said to be more susceptible to it. The symptoms are a feeling of fullness in the head, deafness, tinnitus, vomiting, delirium with visual hallucinations and haemorrhage from the nose, kidney and bowel. Very large doses sometimes produce extreme dyspnoea resembling the air hunger of diabetic coma, together with slowing and weakening of the pulse. I have never seen these bad effects in children, when the dose recommended for acute attacks is given combined with double the quantity of sodium bicarbonate.

In cases where sodium salicylate upsets the stomach it is sometimes necessary to substitute one of the other members of the salicylate group. Of these salicin is less likely to cause unpleasant symptoms, but is less powerful. It may be given in 5 -20 gr. doses with an equal quantity of sodium bicarbonate every two or three hours and gradually diminished. I have found salol (gr 5 - 10), combined with bismuth in a mixture, useful in cases of vomiting due to sodium salicylate, and also in the attacks of diarrhoea with foul smelling stools which sometimes occur in rheumatic children.

LOCAL TREATMENT.

Blistering over or near the affected joints was first advocated by [33] Davis with a view to aiding in the elimination of "morbid matter". There is no reason to believe that it arrests either cardiac or joint inflammation. In the rare cases of pericardial effusion in children blistering is of some service in promoting absorption. Similarly in cases of arthritis where the effusion persists, blistering may be used with advantage.

The application of cold compresses or iced bags was first recommended by [33] Esmarch and Stromeyer, and is still frequently used in cases of pericarditis in which it is believed to check inflammation, hinder effusion, aid in absorption and diminish dilatation of the heart. In acute pericarditis it certainly seems to relieve pain and distress. Hot fomentations and poultices are troublesome and do not have any appreciable effect on cardiac inflammation, but they may relieve severe and persistent pain in the joints. Sodium bicarbon-
-ate fomentations to joints relieve pain sometimes.

Leeches are permissible only in extreme cases of carditis, especially if there is much dilatation of the right side of the heart, when one or two may be applied to the praecordium. Children do not stand the loss of blood well and the fear

inspired by the application of leeches may be harmful to a nervous child.

A useful local application is Methyl Salicylate or Oleum Gaultheria 1 drm in an oz of Lanolin. The drug thus applied is absorbed directly through the skin as can be proved by the appearance of salicyluric acid in the urine, and probably reaches the focus of infection in larger quantities than when it is taken by the mouth. It should be applied distally to the joint as it is absorbed by the ascending lymphatics and so may be carried up past the joint.

Serum Therapy.

[33]Menzer prepared a serum by inoculating horses with living cultures of streptococci from rheumatic sore throats. In cases of rheumatic fever he recommends daily injections of 5 - 10 c.c.s up to a total dose of 50 c.c.s. with intervals of one or two days. He claims that the results of its use in a large number of cases were better than those of any other method, also that it reduces the frequency of cardiac manifestations and relapses, and lessens the likelihood of the development of chronic rheumatism. It is said to be contra-indicated by pericarditis, pleurisy and high fever, but not by endocarditis. There has, however, been

no experimental proof that the serum has the bacteriolytic property which is claimed for it, and other observers have failed to confirm the conclusions of Menzer.

Vaccine Therapy. has been extensively tried in this country, generally with disappointing results. This lack of success may be explained by the fact that acute rheumatism is a disease which does not protect against itself. In fact one attack seems to predispose to further attacks.

Tonsillectomy.

It has not been proved that the tonsils are the usual path of infection in rheumatism in spite of the frequent occurrence of tonsillitis in rheumatic subjects, or else tonsillectomy would take its place as an important prophylactic measure. Moreover it has been found that tonsillectomy in patients who have had one or more attacks of rheumatism does not always prevent further attacks. One reason for this probably is that the infection in the throat is not usually confined to the tonsils but invades the surrounding tissues. However, provided there is no acute attack and the condition of the child's heart will allow it, diseased tonsils should always be removed. By so doing at least one of the foci which may be the source of relapses is removed.

Treatment of an Attack.

In a case of acute arthritis the patient is put to bed between blankets. The bowels are opened with a purge and a daily motion is ensured afterwards. Treatment with sodium salicylate is commenced at once as described above. The salicylate should be combined with twice the amount of sodium bicarbonate to avoid unpleasant symptoms. Locally it is seldom necessary in children to do more than wrap the affected joints in cotton wool or flannels. In the few cases in which the pain is severe or persistent, Methyl salicylate or gaultheria ointment spread on white lint and covered with waterproof material and cotton wool is very useful. While the symptoms are acute the diet is strictly limited to milk and clear fluids. The patient is encouraged to drink freely to aid in the elimination of toxins. Meat extracts are not allowed. When fever and pain have subsided a generous allowance of milk puddings, custard, vegetable broths, cornflour, stewed fruit and bread and butter may be added. But as a too rapid return to a full nitrogenous diet seems to be responsible for a relapse in some cases, I withhold fish and eggs until a week after the temperature has become normal. Meat is not allowed until a week later and is then only given sparingly while the patient is still in bed.

A sufficiently long rest in bed is of the utmost importance. In the absence of all complic--ations I keep the child in bed for at least three weeks after the temperature has become normal. He is then allowed to get up for an hour the first day and a little longer on each succeeding day until he has a full eight hour day out of bed at the end of about a week. Care should be taken against over-exertion in the early stage of convalescence, as this may cause a relapse. Anaemia wasting and weakness require treatment and are best met by tonics such as Parrish's Food, malt extract with cod liver oil and Easton's syrup. This may with advantage be commenced at the end of the third week of rest in bed.

Fresh air is of great importance during convalescence, but chills and damp should be specially guarded against. Where possible the child should be sent away from the environment in which it contracted the disease for three months or more, provided it can have constant supervision. For this purpose the ordinary convalescent homes are generally held to be unsuitable, as the child has too much freedom and excitement there and is likely to over-exert itself. Sending it away to relations in the country is open to the same objections. Special country homes where proper nursing and supervision

are available for such cases are unfortunately very rare in this country, but a few do exist. It is to be hoped that the number of these will rapidly increase in order to meet this very genuine need.

Where the child cannot be sent away and its home is obviously unsatisfactory an attempt should be made to get the family into a more suitable house. Unfortunately, in these days of housing shortage, that is often a very difficult matter. In any case whether the period of convalescence is spent at home or away from home it should always be long.

If endocarditis or pericarditis develops during an attack I continue to give sodium salicylate combined with sodium bicarbonate and make no change in the dosage. If vomiting occurs I stop sodium salicylate for a time and substitute salol combined with bismuth. Cardiac tonics such as Tr Digitalis (mm 5 - 8) and Tr Nux. Vom.(mm 2 - 4) are often given in cases of acute carditis, but, with the exception of Tr Nux.Vom. as a general tonic, it is doubtful whether or not they benefit the patient. Brandy given in 10 - 20 mm doses well diluted with water every 2 - 4 hours should be reserved for severe cases, when there is a tendency to fainting and the pulse is very feeble. Liq. Strychnine (mm $\frac{1}{2}$ - 1) hypodermically every 3 hours may be given at the same time. If cardiac distress causes sleeplessness

the most valuable drug is opium given in the form of pulv. ipecac. co. (gr 2 - 4) three times a day. It often acts like a charm relieving pain and procuring sleep which is invaluable at a critical stage in a severe carditis.

For local pain and distress I have not found it necessary to use the more drastic measures such as leaching and blistering. An ice bag to the praecordium is most soothing. I have recently used antiphlogistin in a case of pericarditis with considerable relief to the patient, the methyl salicylate contained in it probably being largely responsible.

The question of the length of the period of rest in bed necessary in a case of rheumatic carditis has been much debated in recent years. It is generally agreed that the child should be kept in bed until all evidence of activity of the infection has been absent at least two weeks. In the most favourable cases this desirable result may be achieved in about 6 to 8 weeks, but very often several months are necessary and so great an authority as Sir James MacKenzie[34] recommends as much as 6 months rest in bed while there is still infection of the heart. It is often exceedingly difficult, however, to judge whether all infective activity has ceased or not. The following clinical tests have been suggested for guidance in this matter:-

1. The disappearance of anaemia. This, however, is often slight and cannot be relied on.

2. The return of the pulse to the normal is probably the surest evidence that infection has ceased. But, on the other hand, a rapid pulse does not necessarily mean that infection is still present. The increased rate may be due to loss of cardiac tone caused by lack of exercise, or to anaemia even although the infection has ceased. The digitalis test has, therefore, been suggested to give further information. Digitalis is given in large doses for two or three days and its effect on the pulse rate noted. If it slows, the pulse infection may be considered to have stopped and the patient may be allowed to get up. If it does not, infection is still present. This test is based on the fact that while the heart is stimulated by the toxins of infective conditions such as Pneumonia, Pulmonary Tuberculosis, or Rheumatic Fever, digitalis does not slow it. The return of the normal respiratory variations of the pulse is another helpful sign.

3. Disappearance or stabilization of the physical signs. While there is pericardial friction, a changing murmur, and variations in the positions of the borders of the heart, the patient must be kept in bed. On cessation of infection, pericardial friction and associated myocarditis stop, the cardiac borders return to their normal position, or, at any

rate, take up fixed positions, and the diastolic murmur in the mitral area, if present, disappears or ceases to vary its position in the cardiac cycle. All these signs, however, may be absent and yet the heart may be seriously infected. The physical signs by themselves cannot, therefore, be looked upon as a reliable guide.

4. The patient loses his toxic appearance when infection ceases.

5. The temperature is an uncertain guide because serious cardiac infection may exist without a rise in temperature.

6. A gain in weight may assist in coming to a decision, but loss of weight may be due to causes other than rheumatism.

No single test can be relied on. A careful consideration of all the evidence is necessary to arrive at a correct decision.

The change from absolute rest in bed to the stage of convalescence should be brought about gradually. The patient is allowed to get up for increasing periods on successive days, a careful watch being kept on the heart and pulse. Carefully graduated exercises may be commenced after he has been up for three or four weeks. A convalescence of several months is necessary, during which constant care and supervision either at home by the private

doctor, or, better still, at a special convalescent home for rheumatism, should be insisted on. At this stage digitalis given as for adult heart disease is occasionally useful. The least sign of a return of infection should call for a further period of rest in bed.

After the period of convalescence there remains to be considered the question of the child's return to school, and of the amount and nature of the exercise and recreation which he may safely be allowed to undertake. It is probably safer for it to attend school as a day scholar than as a boarder for then, early signs of relapse are less likely to be missed.

The question of exercise is a difficult one which calls for much judgment. Each case should be treated on its merits. Restriction from all games is not necessary in a large number of cases. The heart muscle requires exercise to keep efficient just as much as the skeletal muscles. Further, we must bear in mind that it is repeated infection rather than mechanical defects which ultimately tends to prove fatal in rheumatic children.

The response of the patient's heart to effort will have been guaged to a certain extent during the convalescent stage and will be a guide in choosing the form of his recreation. If the heart

has been badly damaged or the lesion is of a chronic progressive type the more active sports, such as football, swimming and tennis, should be forbidden.

Strict supervision must be kept on the child for the rest of its childhood. Its general nutrition should be kept up to as high a pitch as possible, special care being necessary in tiding it over the dangerous period of puberty. If tonsillitis is present during an attack of acute rheumatism the tonsils should be painted three or four times a day with:

R Salol 33s
Glycerin 3ii
Spr Vini Rect ad 3i

If they are permanently enlarged or diseased after an attack they should be removed provided the condition of the heart will allow of an anaesthetic being given.

Skin manifestations are but little influenced by salicylates. Purpura and urticaria may develop while the patient is taking large doses of sodium salicylate and, indeed, in rare cases both these eruptions may be actually caused by the drug. Purpura tends to get better by itself and does not require treatment beyond rest in bed, although various styptics such as Calcium Lactate, Turpentine and Adrenalin have been employed. The itching of urticaria is relieved by the local

application of 5% Ichthyol in lotion or ointment.

Erythema Nodosum is said to be benefited by sodium salicylate. I give this drug in full dose to combat the infection, but I do not believe that the course of the eruption is much influenced by it. Recently a child under my care who for four days had been taking large doses of sodium salicylate for erythema nodosum on both legs developed a fresh crop on the right elbow and upper arm. I find methyl salicylate ointment applied as for the joints useful for relieving the pain. The most important part of the treatment, however, is rest.

Rheumatic nodules I am convinced are unaffected by large doses of salicylates. Local applications of methyl salicylate also have no effect on them. Their presence serves to warn us of the gravity of the associated cardiac lesions, to the treatment of which all attention should be directed.

Chorea.

In mild cases the patient may recover perfectly, if treated by rest, both mental and physical, and diet only. In severe cases complete isolation with a firm nurse is desirable, but in general practice in an industrial area where this is often impossible, one has to rely on some sensible woman in the family to do the nursing.

The child should be soothed and mildly amused, but allowed no games or books. Hot packs are sometimes more useful in procuring sleep than drugs. Other children should be kept away as much as possible as they cause excitement and annoyance by mimicking the movements. In the presence of cardiac complications rest in bed should be prolonged for several months or as long as there is any sign of infective activity. In an uncomplicated case the patient may be allowed to get up for a few hours a day when the symptoms begin to subside, but care should be taken against over-exertion and chills, and a long rest should always be taken in the middle of the day.

The diet if there is fever or a severe complication should at first consist chiefly of milk, but in the absence of these it may be rapidly increased, milk puddings, fish, eggs, and bread and butter being allowed, as it is not wise to underfeed these cases.

Drugs, although not curative, are often helpful in the treatment of chorea. A purge such as calomel should be given at the commencement and after that a daily motion should be ensured. Salicylates are of value only if there is pyrexia, arthritis, tonsillitis or carditis associated with chorea.

Sodium salicylate combined with sodium bicarbonate will clear up any of the first three and will favourably influence the course of the carditis. It may modify the attack of chorea, but as that is mainly the result of a rheumatic infection, it will continue even after the infection has subsided.

Insomnia and violent movements often require sedatives. Of these Aspirin, Bromides, Chloral, Trional, Phenazonum and Chloretone have all been found useful. Chloral (gr 7) is useful for procuring a night's sleep. Potassium Bromide may be given over longer periods and is often advantag--eously combined with Liquor Arsenicalis.

Arsenic is commonly given as a nerve tonic. It is best given in increasing doses commencing with 2 mms three times a day, and increasing by 1 mm. every third day until 10 mms. are given three times a day. This dose may then be continued until the movements become quieter and then gradually reduced. If arsenism sets in at any stage the drug should be stopped at once . In Chorea Paralytica stimulants are required. In Chorea insaniens sedatives may have to be pushed as there is danger of exhaustion.

During convalescence massage and exercise are beneficial. Massage is specially useful in the paralytic type of case in which it may be begun at an earlier stage than usual. Exercises should be simple at first, but later special games requiring

careful voluntary control such as knitting, exercises on the piano and building castles with bricks should be tried. All cases should have cod liver oil and malt for wasting, and iron for anaemia. A convalescence of three months in uncomplicated cases, and six months in those with cardiac complications, preferably in the country, should be insisted on.

Defective eye-sight, adenoids and other sources of peripheral irritation should be corrected and the parents warned against sending the child in for examinations or subjecting it to nervous tension or worry for some years.

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SUMMARY.

1. Rheumatism in children is a serious disease because of its special affinity for the heart. The systematic study of the disease has been shelved until comparatively recent years. In order to clear up the etiology and improve our treatment of the disease it is necessary that the profession as a whole realizes its gravity and takes active steps to fight it.

2. None of the older theories on the cause of rheumatism such as the chemical, humoral and nervous theories will bear examination. A weight of evidence has been collected within recent years to prove beyond reasonable doubt that the specific cause is bacterial. Although the identity of the organism responsible is still in dispute the bulk of evidence is in favour of a streptococcal origin of the disease. There are, however, still some difficulties with regard to the differentiation of the particular strain of streptococcus which calls for special investigation by a body of skilled bacteriologists.

3. Important predisposing causes are age, climate, poverty, damp dwellings, damp clothes and heredity. The relative importance of these, however, is still in dispute and mush work has yet

to be done before we can apportion blame correctly.

It is in this branch of investigation that general practitioners and school medical officers can be of great assistance. I should suggest that local medical societies such as that of Blackburn (which has amongst its members public health officers, the local infirmary staff and general practitioners) appoint committees of investigation to gather information on this subject from all the practitioners and the school medical officers in the area.

If this scheme were carried out both in urban and rural districts in different parts of the country our knowledge of the disease would be greatly increased and we should be better able to cope with it.

4. The main route of infection in rheumatism is probably through the tonsils, but the exact relationship of tonsillitis to rheumatism is not yet defined. Attacks of rheumatism in children with healthy tonsils or in children whose tonsils have been removed suggest the probability that the causal organism has other ways of entering the body. A possible route of infection is through the bowel, but apart from the increased streptococcal content of the faeces in a large percentage of cases, we possess very little evidence in support of this.

5. In children rheumatism is an insidious disease characterized by many and varied manifestat--ions, a marked tendency to relapses and recurrences and a special affinity for the heart. In its insidious attack on the heart lies the danger of the disease, hence the importance of recognising and dealing with early symptoms such as growing pains, headaches, fibrositis, slight sore throats, listless--ness and anaemia.

6. The heart is liable to suffer in all or more than one of its tissues at the same time, especially in subacute progressive cases. First attacks on the heart are represented clinically by the dilatation of acute myocarditis, by acute endocarditis, and much less commonly by acute pericarditis. In progressive or relapsing cases we meet with chronic myocarditis, mitral stenosis and subacute pericarditis. Repeated infection of the myocardium plays a more important part in the heart failure of early life than does valvular disease.

More prolonged investigation of a large number of cases is necessary in order to explain the occurrence of presystolic murmurs in middle and late life in people who have had no cardiac symptoms and who cannot give a history of rheumatism.

In the rheumatism of children the joints suffer much less than in that of adults. Muscular and periarticular fibrous tissues are more commonly

involved. The nervous system is specially liable to attack.

7. In treatment, prevention is our chief hope in reducing the incidence of cardiac rheumatism. Special attention should be directed to housing and guarding rheumatic children against damp and chills.

8. The salicylates are the most useful drugs we have in the treatment of rheumatism. Combined with sodium bicarbonate they may be given in large doses in all rheumatic manifestations. They appear to act specifically in the acute stages of the disease but cannot remedy structural damage when it has occurred. Their usefulness in preventing cardiac inflammation is largely discounted by the insidiousness of the attack on the heart.

9. Proper after-care of the rheumatic subject will greatly help in preventing relapses and recurrences and will prolong the lives of cardiac cripples. For this the number of special cardiac homes should be rapidly augmented.

Tonsillectomy lessens the tendency to recurrences but does not prevent them altogether.

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Violet E. - Female, aged 18. Photo of hand showing rheumatic nodules along extensor tendons.

Violet E. Female. aged 18. Photo of elbow showing rheumatic nodules.